BRITISH MEDICAL JOURNAL

LONDON SATURDAY JANUARY 29 1944

LOCALIZED SUBCUTANEOUS OEDEMA WITH WEAKNESS OF LIMB MUSCLES

A SYNDROME DUE TO POLYARTERITIS NODOSA

BY

RONALD MacKEITH, D.M., M.R.C.P.

Temporary Surgeon Lieutenant, R.N.V.R.; Medical Specialist, Royal Naval Medical Service

The three cases of polyarteritis nodosa described here are of interest for several reasons. They all showed as the two salient features a localized soft pitting oedema of the skin of one or more limbs together with asymmetrical muscular weakness—a clinical picture not previously described. Two of these patients recovered from the illness. All three cases were diagnosed in life, the diagnosis being confirmed by biopsy. The three cases were seen in one hospital in a period of 18 months, which suggests that Leishman (1937) was right in suggesting that this disease, though very varied in its forms, should be recognized more frequently. It may be observed that during this period, although polyarteritis was being looked for, in only one other case was it suspected, and this showed a clinical picture very similar to that of the three cases. The biopsies, however, were negative.

Case I (an Engineer aged 27)

The family history was irrelevant. There was a previous history of a cough for 12 months and of a coryza towards the end of April, 1940. On May 1, 1940, while at work, the patient injured his right calf and had to take to bed. Two days later, while in bed, he found that he had great difficulty in moving the right thigh—attempts causing pain in the groin—and that he was unable to lift his right forearm off the bed or to raise the arm at the shoulder.

On May 6, 1940, he was admitted to St. James Hospital, London. He was of slim build and in fair general condition; his temperature was 99.6° F., the pulse rate 88, and the respiration rate 24. No abnormal signs were found in mouth, lungs, heart, or abdomen; his blood pressure was 135/85 (a week later it was 120/80). A mild degree of clubbing of the finger-nails was present. There was considerable weakness of the right deltoid and biceps muscles, which were tender on palpation. The right upper arm was swollen—the circumference being 9½ in., against 8 in. on the left side—from diffuse soft pitting oedema. The skin was not inflamed. There was no sensory loss or any paraesthesiae at this time or later. The muscles of the right thigh were weak and tender, and pain was felt on externally rotating the flexed thigh. There were two subcutaneous nodules on the outer side of the right calf, but no oedema there. The left arm and leg were normal. The patient was treated by rest in bed and a high vitamin diet (with bemax, cod-liver oil, ascorbic acid, and iron), and massage at a later stage.

Investigation on Admission.—Urine, nothing abnormal. Blood urea, 36 mg. per 100 c.cm. Blood count: R.B.C. 5.5 millions; Hb 92% (Haldane); platelets normal; W.B.C. 12,000—neutrophils 79%, lymphocytes 15%, monocytes 6%. (Nine days later: Hb 97%, W.B.C. 12,000, neutrophils 69%.) Sedimentation rate, normal. Sputum: no tubercle bacilli on 3 examinations; culture pneumococci. Radiograph of chest (May 13, 1940): Increased markings at both hila, and a small amount of fluid or thickened pleura at the left base. (A further chest radiograph two weeks later showed no change.) A radiograph of the right upper arm revealed no abnormality. Cerebrospinal fluid: Clear, colourless, 2 cells per c.mm.; protein, 40 mg.; globulin *nil*; chlorides, 740 mg.; W.R. and Lange negative. Blood W.R. and K.R. negative. An electrocardiogram on May 26 showed poor T-waves and slight elevation of ST_1 with depression of ST_3. These changes were still present 10 days later.

Progress in Hospital.—For 2½ weeks he had a symptomless pyrexia and a rather longer-lasting tachycardia (Fig. 1). The swelling of the right arm had increased the day after admission to 10½ in. circumference, but in a few days it began to diminish and in two weeks it was gone. The weakness of the right arm and shoulder persisted for 10 days before improving, but had cleared up in 4 weeks. A week after admission there was still tenderness at the insertion of the biceps muscle, and some pain was felt on using the triceps. There was also a tender nodule, about 0.5 cm. in diameter, beside the brachial artery at the level of the biceps tendon. The power of the right thigh improved from the time of admission. A week after admission one small tender subcutaneous nodule was observed on the outer side of the right calf, and also an erythema-nodosum-like lesion of the skin over the shin. On May 11 he complained of pain in the left orbit, but no lesion or oedema was seen. The patient started getting up on June 3, and for a few days after this the dorsum of the right foot showed a little soft oedema. There was no tenderness or weakness of muscles of the legs, and the ankle-jerks were present though not brisk. On June 9, five weeks after admission, he left the hospital quite well.

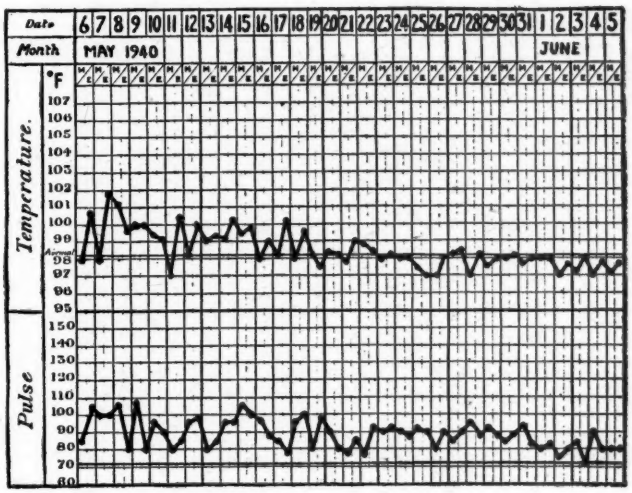


FIG. 1.—Temperature and pulse chart of Case I.

On May 22 a biopsy specimen was taken of a nodule, 0.3 cm. in diameter, attached to a small subcutaneous vessel in the right arm. The histological report was: "Polyarteritis nodosa in nodule from subcutaneous tissue of arm."

Two and a half years later, in Oct., 1942, he and his private doctor both reported that he had remained perfectly well.

Case II (a House-painter aged 63)

This man had had a chronic cough for many years. He took alcoholic drinks in moderation only. On Nov. 5, 1940, he developed a "numbing pain" in the lumbar region and shoulders radiating down the arms. There was no weakness of the limbs. Two days later his ankles began to swell and he became short of breath. On Nov. 11 he was admitted to St. James Hospital.

On admission his general condition was fair. His temperature was 97.6° F., pulse 84 regular, and respirations 20. The heart was normal in size and sounds; the brachial arteries were thickened. There was soft pitting oedema + + of the ankles, but nothing else to suggest congestive heart failure. He was not orthopnoeic. The lungs showed signs of diffuse bronchitis, but there was little sputum and no haemoptysis.

Investigations on Admission.—The urine was normal. There were moderate anaemia and a leucocytosis (R.B.C. 3.5 millions, Hb 77%; W.B.C. 15,200—neutrophils 85%, eosinophils 1%, lymphocytes 11%, monocytes 3%). The blood W.R., K.R., and gonococcal complement-fixation test were negative. The blood sedimentation rate was very rapid—53 mm. on 100 mm. column at 1 hour. The sputum showed no tubercle bacilli; one culture yielded a fair growth of Friedländer's bacillus. An *x*-ray film of the chest showed only the increase in vascular markings common in chronic bronchitis. Radiographs of the limbs revealed no abnormality in bones or joints. The blood urea six weeks after admission was 60 mg. per 100 c.cm., and

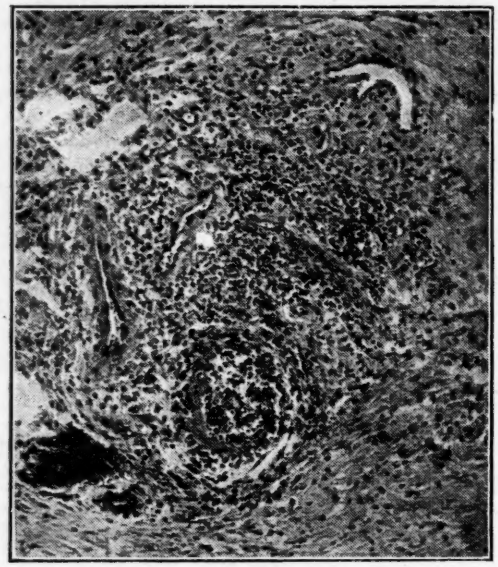


FIG. 2.—Case II. Photomicrograph showing necrosis of arterial wall. Stained haematoxylin-eosin. (× 95.)

the plasma protein at this time showed a great rise in albumin. Total protein, 9.9% (normal 5.6 to 7.8%): albumin 7.55% (normal 3.4 to 4.9%); globulin 2.35% (normal 1.2 to 2.9%).

The patient was treated by rest in bed, with bemax, iron, and theophylline by mouth and simple analgesics for the pains.

Progress in Hospital.—Four days after admission he developed pain in the legs. The calf muscles were tender and movements of the ankle-joints were painful and weak. The ankle-jerks were absent, the knee-jerks faint, the plantar responses flexor, and the abdominal reflexes present. There was no pain or oedema in the upper limbs at this stage, but the triceps-jerks were absent. No inflammation of the skin or any nodules were present at this or any later stage.

Though the oedema of the feet persisted, the pain in the legs cleared up in a week; but before it had gone pain developed at both elbows. On Nov. 17 neither elbow could be fully extended; the right ulnar nerve was tender but not the left. In contrast there was pitting oedema of the dependent skin at the left elbow, but none on the right. There was also tenderness deep in the right antecubital fossa. A blood count at this time showed no leucocytosis (W.B.C. 6,200), though the B.S.R. was still very rapid—48 mm. at 1 hour. On Nov. 23 pain and soft pitting oedema had appeared at the back of the left thigh, and the pain at the left elbow was worse.

On Nov. 27 the patient complained of paraesthesiae in the right hand; but the legs and arms were much more comfortable, and the swelling at the left elbow began to diminish. A week later, however, he had a severe pain in the left forearm, with tinglings in the left hand, diffuse swelling of the left forearm, and weakness of the left wrist and hand. On the right side there was now no weakness or pain.

On Dec. 6 and 7 he complained of a "different sort of pain" in the abdomen. There was tenderness at the renal angles, and the urine, previously normal, contained albumin, with many red cells and a few leucocytes in the deposit. Some albumin and blood were present in the urine every day from this time till his death five weeks later; and in the last three weeks of his life there were increasing numbers of granular casts. On Dec. 6 it was observed that a mild clubbing of the finger-nails had developed since his admission.

On Dec. 9 he had a severe pain in the left leg and tenderness in the lateral part of the left popliteal fossa, but no fresh oedema. A week later he was becoming drowsy and losing ground. There was now oedema of the ankles, the back of the left thigh, and the left upper arm. The blood count on Dec. 17 showed Hb 72%, W.B.C. 15,600 (neutrophils 80%, eosinophils 5%). He was given a transfusion of a pint of citrated blood, and two days later the blood count was Hb 80%, W.B.C. 15,600 (neutrophils 84%, eosinophils 4%).

He was now (Dec. 19) complaining of severe pain, and the oedema was present in the right thigh as well as the left, and there was a manifest left wrist-drop. He was started on injections of vitamin B_1,

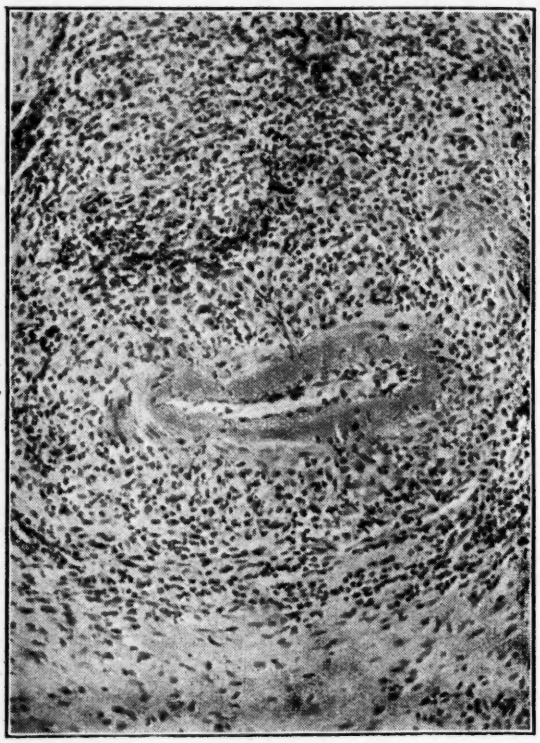


FIG. 3.—Case II. Photomicrograph showing polymorph infiltration in vessel wall. Stained haematoxylin-eosin. (× 140.)

2 mg. on alternate days. On Dec. 24 he was definitely better, with less pain and oedema but some mild paraesthesiae of the hands and feet. The knee, ankle, triceps, and supinator jerks were absent, and the responses from the biceps were faint. The blood pressure was 150/110; there were no signs of congestive heart failure.

The anaemia and leucocytosis were both increasing (Hb 66%; W.B.C. 22,000, neutrophils 97%, on Jan. 3, 1941). He became steadily weaker, his face getting thinner, with an earthy pallor. Oedema now appeared in the right as well as the left arm and increased, and in the legs it became generalized. A course of sulphapyridine was without any good effect. He died on Jan. 12, 1941.

The biopsy report on material taken on Dec. 9, 1940, from subcutaneous tissues and flexor muscles of the left arm was: "Polyarteritis nodosa. Hyaline necrosis of wall of small artery with surrounding acute purulent inflammatory reaction." (Figs. 2 and 3.)

Necropsy.—A necropsy was made the day after death. The bronchi contained much muco-pus and were somewhat dilated at their terminations. Both lower lobes showed diffuse bronchopneumonia. The heart muscle appeared quite normal. There were lines of redness along the descending branches of the right coronary artery, but no nodules or aneurysms. Atheromatous patches were present in both coronary arteries and in the aorta. No nodules were seen on the mesenteric vessels. The liver showed only slight venous congestion. Both kidneys had a smooth surface mottled from many

cortical infarcts. On section the cortex was of normal width; there were two small circular areas suggestive of aneurysm.

Histological Report.—The kidneys showed diffuse chronic nephritis and focal ischaemic nephritis, and also severe chronic purulent inflammation and necrosis of arcuate arteries, some with canalized organized thrombosis. Aneurysmal dilatation of an artery and a purulent infarct of the cortex were also seen. There was chronic purulent inflammation and necrosis of coronary arteries of the heart and of arteries in the mesentery. Tissue from the axilla showed chronic purulent inflammation of the axillary artery and of its branches in adherent nerve and muscle. Changes in other organs were bronchopneumonia, chronic septic spleen, congestion of the liver, and fibrosis of the myocardium.

Case III (an Escalator Engineer aged 58)

This patient had a history of recurrent coughs. He took no alcoholic drinks. About August 10, 1941, he developed aches and pains all over and a dull pain at the navel. There were night sweats, and he noticed that he was passing less urine. He had an occipital headache, poor vision and weeping eyes, and some attacks of faintness. His appetite remained good. A week after the onset of these symptoms he began suddenly to have numbness and tinglings and weakness of the hands, progressing to uselessness, and similar paraesthesiae and a slighter degree of weakness of the legs and feet. Two weeks after the illness began the left side of his face became puffy and the ankles swelled. He was admitted to St. James Hospital on Sept. 1, 1941.

He was in fair general condition, of sallow complexion; temperature 98°, pulse regular 76, vessel not thickened; respirations rapid (44) but not distressed. The heart size and sounds were normal. There was no orthopnoea; the lungs were clear. There was no venous engorgement or hepatic enlargement; his blood pressure was 200/110. Slight tenderness was felt in the loins. The urine contained much albumin, the deposit showing many red cells, a few leucocytes, and some hyaline casts; no organisms were seen or grown on culture. There was much soft pitting oedema of the upper limbs, especially in the dependent part of the forearms and near the elbows; this was more extensive on the right side. There was also soft oedema behind the ankles, especially on the left. The face was free of oedema.

Examination of the nervous system revealed no defect of the cranial nerves. Both deltoids showed some weakness. On the right side there was slight power of flexion of the fingers but complete wrist- and finger-drop, and flexion and extension of the elbow were weak. On the left side he could not oppose the thumb and fourth finger, but flexion and extension of fingers and of wrist were good; there was fair power of flexion of the elbow but considerable weakness of extension. The lower limbs showed slight general weakness. The right triceps, biceps, and supinator jerks were absent, while on the left they were respectively sluggish, normal, and sluggish. The left knee-jerk was brisker than the right; the ankle-jerks were both sluggish. The plantar responses were flexor, the lower abdominal reflexes absent, and the upper normal.

The electrical reactions of the muscles were tested. In the left upper limb these were normal. On the right all muscles reacted to galvanism; to faradism the triceps and extensor muscles of wrist and fingers did not react; the deltoid and biceps reacted weakly; and the muscles of the flexor aspect of the forearm and the intrinsic muscles of the hand reacted normally.

There was on the right a gross loss of sensation of the hand and lower part of the forearm; and on the left, of the hand only. The legs showed diminished sensibility to light touch, of stocking distribution. Position and vibration sense were good. There was no tenderness of the muscles of the upper or lower limbs.

The combination of an asymmetrical polyneuritis with oedema suggested the probability of polyarteritis nodosa. Dr. E. A. Blake Pritchard saw the case; he thought it was not a syndrome due to peripheral neuritis. The patient was treated by rest in bed with a high vitamin diet. He was given aneurin 10 mg. by intramuscular injection daily for four days, and splints and physiotherapy for the limbs.

Investigations.—Circulation times (Sept. 12) normal (ether 8 and 10 seconds; cyanide 15 and 15 seconds). An *x*-ray film of the chest showed no evidence of disease of the lungs, heart, or mediastinum. Blood W.R. and K.R., negative. Blood urea variable—70 mg. per 100 c.cm. (Sept. 4), 36 mg. (Sept. 16), 70 mg. (Sept. 23). Plasma proteins (Sept. 16) were very high from excessive amount of albumin (total 8.45%—albumin 5.9, globulin 2.55; albumin/globulin ratio 2.3 : 1 (normal values: total 5.6–7.8%; albumin 3.4–4.9, globulin 1.2–2.9; albumin/globulin ratio 2–3 : 1). A blood count on admission (Sept. 2) showed anaemia and a neutrophil leucocytosis. (R.B.C. 4.3 millions, Hb 68%, C.I. 0.8; group O; W.B.C. 9,800—neutrophils 84%, eosinophils 4%, lymphocytes 10%, monocytes 2%.) Serum salts (Sept. 23): chloride 580 mg., potassium 14.5 mg., sodium 324 mg. (normal values: 560–620, 16–20, 325–350).

Progress in Hospital.—During his 3½ months in hospital the patient's temperature was often raised (Fig. 4). A course of sulphathiazole started on Sept. 13 had no effect. The blood pressure —200/110 on admission and 225/135 two days later—gradually fell to 160/90 (Oct. 23). The abnormal urinary constituents gradually diminished to a little albumin with a few red cells in the deposits. The oedema varied but never disappeared. There were never any signs of heart failure, right- or left-sided. The pain in the limbs varied; at one time he complained of shooting pains, but chiefly of the numbness and progressive weakness of the hands. The anaemia increased; three weeks after admission the Hb was 48%, with W.B.C. 7,200 (neutrophils 79%, eosinophils 7%).

On Oct. 1, a month after admission, he developed diarrhoea. The stools contained no mucus, blood, parasites, or pathogenic organisms.

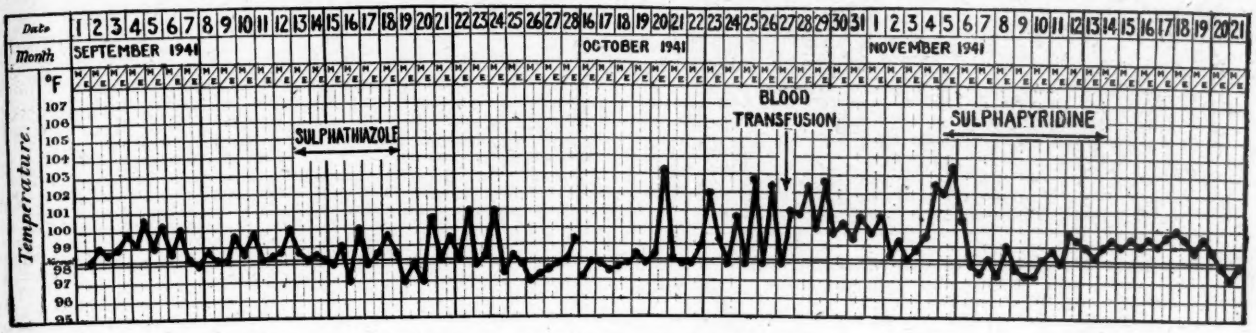


FIG. 4.—Temperature chart of Case III.

Per rectum several small submucous nodules could be felt. Sigmoidoscopy showed a normal mucosa. By the middle of October there was much soft oedema in the lumbar region as well as in the elbow regions and at the ankles. The muscular weakness had increased in the left wrist, in both arms, and in the dorsiflexors of the ankles. There was marked wasting of the intrinsic muscles of the hands and of all the limbs. The loss of pinprick perception in the hands was more extensive. In the feet there was analgesia on the right to the midfoot and on the left to the ankle.

On Oct. 23 the pyrexia became more severe without evident cause. On Oct. 27, as the anaemia and leucocytosis persisted (Hb 56%, W.B.C. 10,200—neutrophils 81%, eosinophils 2%), he was given a blood transfusion. During the following week he developed a cough and fever, with diarrhoea and signs of consolidation of the right lower lobe (Nov. 5). He was given a course of sulphapyridine, and the fever and diarrhoea cleared up. For a time he seemed to be losing ground, but at the end of November he was feeling better, the oedema was lessening, and no eosinophils were seen in a blood count (Hb 58%, W.B.C. 8,600—neutrophils 74%). The chest still gave many moist sounds. He continued to improve, though the hands and fingers were becoming contracted despite splinting and so on. At his own request he went home on Dec. 23, 1941, though he was still too weak to sit up in bed by himself.

Further Progress.—At home he remained in bed for three months, improving steadily. Six months after leaving the hospital he came to the out-patient department. His face was fat and ruddy; there was no oedema anywhere. Both hands were wasted and contracted —on the left a claw-hand, but on the right flexion at the metacarpophalangeal joints. The power in the upper arms and elbows was fair. The blood pressure was 245/150, and he had some morning headaches and giddiness. In Oct., 1942, he wrote to say that he was feeling fine; the left hand was still weak and contracted; but the right, which had been the worse, he was able to use for shaving and writing. The legs were still improving, and he could walk a mile or so.

Biopsy Reports.—A week after admission a biopsy specimen was taken of skin, subcutaneous tissue, and muscle from the back of the right forearm. The report on this was: "Slight chronic inflammation of fragment of skin, and fibrosis and degeneration of fragments of voluntary muscle. No evidence of polyarteritis nodosa." A second biopsy was made a month later (Oct. 9, 1941). The report on this was: "Degeneration and slight fibrosis of voluntary muscle. Changes consistent with polyarteritis nodosa in one small artery." (Fig. 5.)

FIG. 5.—Case III. Photomicrograph showing inflammatory reaction in vessel wall. Stained haematoxylin-eosin. (× 190.)

Discussion

Three cases of polyarteritis nodosa, in which the diagnosis was confirmed by biopsy, were seen in 18 months. In a further case, clinically similar, the biopsies were negative. (This was a woman, aged 66, who had had diabetes mellitus and probably hypertension for years. She developed stiffness, pain, and weakness in both forearms, with paraesthesiae in the fingers. Much soft pitting oedema of the forearms occurred, with some contracture of the fingers. There was cardiac hypertrophy, but no evidence of congestive heart failure. She had high plasma proteins; total 7.6% (normal 5.6 to 7.8%)—albumin 5.35% (normal 3.4 to 4.9%), globulin 2.25% (normal 1.2 to 2.9%). After two months the right arm and hand improved; the left remained contracted. Though the clinical picture and the high plasma proteins closely resembled the three cases described below, two biopsies showed no evidence of polyarteritis nodosa; this case therefore cannot be considered as proved to be polyarteritis nodosa.)

The chief features of the three proved cases are summarized in the Table. They showed, as the salient features, localized soft oedema of the subcutaneous tissues of one or more limbs, with asymmetrical muscular weakness. The aetiology of polyarteritis nodosa is still without any definite conclusion, but it is interesting that two of the cases started with aches all over and the other shortly after a coryza. All three showed a leucocytosis, but eosinophilia was not a feature. Skin nodules were present in one case only. (Harris *et al.* (1939) pointed out that eosinophilia and nodules are too rare to be of practical value in diagnosis.) In the two cases in which the estimation was made, the total plasma proteins were very high owing to increase of plasma albumin. This finding may be of diagnostic value. The treatments employed were simple: rest in bed, a high vitamin diet supplemented by injections of vitamin B_1, and splinting and massage for the limbs. The progress of Case I suggests that it is possible to recover completely, while in Case III there was apparently complete recovery from the acute attack, though certain sequelae remained.

Summary of Cases

	Case I	Case II	Case III
Sex and age	M. 27	M. 63	M. 58
Previous history	12 months' cough; recent coryza	Chronic bronchitis	2 years' recurrent bronchitis
Onset	Sudden weakness right arm and thigh	Pains in back and dyspnoea	Aches all over
Oedema	Right upper arm	Successively in ankles, left elbow, left thigh, right elbow, and both legs	Elbows, forearms, ankles, and lumbar region
Nodules	2 in skin of leg	None	In rectal mucosa
Muscular weakness	Right arm and thigh	Left wrist-drop, etc.	Right wrist-drop, etc.
Muscle tenderness	Yes	Yes	No
Contractures of hands	No	No	Yes (late)
Reflexes	—	Impaired or absent in affected limbs	Asymmetrically impaired
Sensory disturbance	None	Paraesthesiae	Paraesthesiae; anaesthesiae
B.P. on admission	135/85	150/110 (five weeks after admission)	200/110
Urinary abnormalities	None	None at first; then albumin, blood, and casts	Albumin ++
Lungs	Thickened pleura (*x*-ray)	Increasing bronchitis	Intercurrent pneumonia
Clubbing of nails	Mild	Mild	None
Anaemia (Hb %)	92%	77%, falling to 57%	68%, falling to 56%; normal six months later
Leucocytosis	12,000 and 12,000	6,200 to 22,000	10,200 to 7,200
Eosinophils %	0, 0	1, 0, 5, 4, 0, 0	4, 7, 2, 2, 0
Plasma proteins (%):			
Total (normal 5·6–7·8)	—	9·9	8·45
Albumin (,, 3·4–4·9)	—	7·55	5·9
Globulin (,, 1·2–2·9)	—	2·35	2·55
Biopsy	Positive	Positive	Neg. Pos.
Course of disease	Recovered in one month. Remains well 2¼ years later	Died after two months' illness	Recovered after six months; with residual contractures of hands and weakness of legs

Summary

Three cases showing localized soft oedema of the subcutaneous tissues of the limbs with asymmetrical muscular weakness are described. The diagnosis of polyarteritis nodosa was confirmed by biopsy. A large increase in plasma albumin was found in the two cases in which this estimation was done. One patient recovered completely, one recovered from the acute illness with muscular contractures and hypertension, and one died.

I thank Dr. W. Allen Daley, Medical Officer of Health to the London County Council, and Dr. N. S. Craig, Medical Superintendent of St. James Hospital, for permission to publish these cases. I wish also to thank Miss E. Edwards, S.R.N., Miss Westlake, S.R.N., Dr. M. L. Playfair, and Dr. A. B. Bratton for their help.

REFERENCES

Leishman, A. W. D. (1937). *Lancet*, **1**, 803.
Harris, A. W., *et al.* (1939). *Arch. intern. Med.*, **63**, 1163

The University Extension and Tutorial Classes Council, in co-operation with the Provisional National Council for Mental Health, will hold this year, provided enough applications are received, the usual course for medical practitioners on mental deficiency and allied conditions. The lectures will be given at the London School of Hygiene and Tropical Medicine, Keppel Street, Bloomsbury, and the date fixed for the course is April 24 to May 5. The University of London will grant certificates of attendance to those who attend regularly, taking both theoretical and practical work. The registration fee, to be paid at the time of application, is 10s. 6d., and the fee for the course is £5 15s. 6d. Cheques should be made payable to the Provisional National Council for Mental Health, and crossed Barclays Bank Ltd. All communications with reference to the course should be addressed to Miss Evelyn Fox, C.B.E., c/o University Extension Department, University of London, 39, Queen Anne Street, W.1. Intending candidates are asked to apply at the earliest possible date, but in any case by March 18. Detailed time-tables and other information will be sent out a week before the course opens to each person proposing to attend. Should the course have to be cancelled all fees will be returned.

EMPHYSEMA OF THE LUNGS*

BY

RONALD V. CHRISTIE, M.D., D.Sc., F.R.C.P.

Professor of Medicine in the University of London; Physician to St. Bartholomew's Hospital

PART II

Physical Signs

The mechanism by which loss of pulmonary elasticity may lead to enlargement of the thoracic cage has already been discussed. The diaphragm, because it is no longer pulled upwards by the lungs, descends and becomes less convex, and its movement on inspiration is greatly diminished. Because of its loss of tone the diaphragm may also present an irregular outline in the radiograph. The intercostal muscles will also be unable to perform their proper function, as they are concerned with moderate inspiration and the chest is already expanded when inspiration begins: to expand the chest further the accessory muscles will have to be used, particularly the pectorals, and these raise the front of the chest as a whole in a "heaving" manner, such as occurs in a normal individual towards the end of a forced inspiration. Thus inspiration has to be performed by means of an unnatural inspiratory effort, and the patient with emphysema will encounter even greater difficulty in deflating his lung. Normally, expiration is largely, if not wholly, a passive act: the thoracic cage is pulled inwards by the elastic recoil of the lung. With loss of elasticity there must be loss of elastic recoil, so that if the lung is to be deflated it has to be squeezed. The respiratory musculature was not built for this task, and the intercostals have to be assisted by the accessory muscles of expiration: the muscles of the abdominal wall can often be felt to contract on expiration, which is prolonged as it is in other conditions, such as asthma and tracheal obstruction, in which the lungs have to be compressed by an active muscular effort. With so extensive an impairment of both inspiration and expiration it is not surprising that the vital capacity and chest expansion are reduced.

Many of the physical signs which can be elicited from the lung itself can be explained on the basis of loss of elasticity. I have already emphasized that, in the inelastic lung, distribution of expansion might be expected to be far from equal and should be greatest where the force is applied, which is on the peripheral parts under the visceral pleura. It would be reasonable to expect that, as the application of this force is repeated many thousands of times daily, the superficial air sacs next the pleura would in time become over-distended. With over-distension their supporting framework would be lost and further stretching would become easier, so that a true vicious circle might be established. It is easy to imagine that this mechanism could be the cause of the over-distension of superficial air sacs, with bulla formation, which occurs in emphysema, although the usual explanation given is that cough distends these air sacs. It is obvious, however, from the mechanism of coughing, which is discussed below, that the lung is compressed rather than distended, and it does not seem possible that cough could be responsible for the formation of bullae, except perhaps in poorly supported areas such as the apex of the lung. Presumably it is this superficial over-distension of air sacs and bulla formation which causes the remarkable hyperresonance of the percussion note—a note which is often more resonant than over a pneumothorax. Why this should occur has not been explained, but it may well be that the over-distended air sac acts as a sound-box or resonating chamber, in the same way as does a cavity. It is also easy to imagine how this distension might occur, more particularly along the edges of the lung, where so-called pleural sinuses exist. With each inspiratory effort on the part of the pectoral muscles the anterior chest wall is pulled outwards and upwards as a whole, and much of this force must be expended on pulling the lung into the pleural sinuses so as to fill the potential gap between the chest wall and the heart. In time this part of the lung will become over-distended, so that there is a gap between the heart and the chest wall, filled by lung. This space can be clearly seen in the lateral view of a radiograph, and it explains a variety of physical signs. The absence of the apical impulse, loss of cardiac dullness, and distant heart sounds necessarily follow. The expansion of the lung, so that it covers the heart in considerable depth, may also in part account for the increase in antero-posterior diameter and the barrel shape of the chest. Similarly the loss of liver dullness must in part be caused by expansion of the lung into the pleural sinuses, although the low position of the diaphragm is of course another factor.

All these changes can be explained directly or indirectly on the basis of loss of elasticity; but it should not be forgotten that enlargement and deformity of the thorax and its contents are partly dependent on other factors, such as the elasticity and mobility of the thoracic cage. These factors vary with the age and build of the patient, and are presumably responsible for the inconstancy and unreliability of the physical signs of emphysema. There remain several physical signs which have not been discussed or explained. Breath sounds are usually faint, especially at the bases, where they may be inaudible; but as I do not know how vesicular breath sounds are produced in health it is difficult to discuss the reason for their suppression. A possible explanation, held by some Continental writers, is that vesicular breathing is produced by the jet of air thrown into the atrium from the alveolar duct. The absence of this mechanism in emphysema has already been discussed, and may be responsible for the suppression of breath sounds. Vocal resonance and fremitus are sometimes decreased in emphysema. I have no explanation to offer for this, as I do not understand the mechanism of conduction of the spoken voice in the healthy lung, although I know of several theories which do not fit the facts.

There are several factors which may contribute to the formation of the barrel-shaped chest, and, although these are not entirely independent of one another, their relative importance probably varies from patient to patient. I have already described how loss of elasticity contributes two factors towards this change: the first was the inspiratory position of the chest, and the second the expansion of the lung between the heart and the sternum. A third important factor must be the dorsal kyphosis, which is distributed throughout the thoracic vertebrae, and which may produce an increase in the antero-posterior diameter by the same mechanism as in Pott's disease. There are thus at least three separate causes for the deformity of the chest, and another factor must be the diminishing flexibility of the chest wall with advancing age. It is not surprising, therefore, that the barrel-chest phenomenon is variable, being marked in some cases and inconspicuous in others.

Thus loss of elasticity alone could directly or indirectly cause the dyspnoea of emphysema, almost all the physical signs, and a considerable proportion of the morbid anatomical appearances. There are, however, a number of changes characteristic of emphysema which I am unable to explain on the basis of loss of elasticity. These are the destruction and degeneration of alveolar walls and blood vessels, the degeneration and atrophy of the pleura, and the degeneration with loss of elasticity of the costal cartilages and the thoracic vertebral cartilages. All these structures are part of the respiratory apparatus, and it is reasonable to suppose that the cause of this widespread damage is also the cause of the degeneration of the elastic fibres themselves. The nature of this destructive agent is discussed below.

Aetiology

In seeking the fundamental cause of emphysema I have described the fallacy of thinking in terms of over-distension and overstretching of the lungs. The lungs as a whole are not overstretched, and we should think rather of some agent which produces destruction and degeneration of many of the structures concerned in respiration, both in the lung and outside it. With this in mind I propose to review briefly the various theories that have been suggested as the cause of emphysema.

The expiratory theory is that the air sacs become over-distended because they cannot be emptied. The inspiratory

* An abridged version of the Goulstonian Lectures delivered before the Royal College of Physicians on March 4, 9, and 11, 1943.

theory claims that over-distension is produced because too much air is drawn into the lungs by the increased inspiratory effort which precedes coughing and which may be associated with asthma, manual labour, and occupations such as glass-blowing and the blowing of wind instruments. There is also a theory that emphysema is primarily due to a faulty position of the chest and that it is the enlargement of the thoracic cage which causes overstretching of the lung. Many criticisms have been levelled against these three theories, but I will only repeat that, from the evidence already described, all of them appear to be based on the false assumption that the lung as a whole in emphysema is truly overstretched and over-distended.

It has been claimed, from quite unconvincing evidence, that local impairment of nutrition is the cause of emphysema. I have pointed out that under resting conditions there is no diminution in the total amount of blood flowing through the lungs, and it is also true that in other diseases there may be more extensive atherosclerosis than is seen in emphysema, although the lung does not become emphysematous. For these reasons it is improbable that the vascular changes in the lung are of any aetiological significance.

It has been suggested that the lung changes are primarily degenerative or atrophic; but this is too easy a way of evading the issue, as it by no means explains the many aetiological factors known to be involved. The most obvious of these concerns respiratory obstruction. Almost all cases of emphysema have a long-standing history of chronic bronchitis or asthma, or both, and this is also true of emphysema in horses and cattle. The effects of cough and bronchial spasm on the lung, and their mechanism, are therefore of primary importance in any discussion on the aetiology of this disease. The mechanism of coughing is simple. There is a short inspiratory effort which by no means fills the chest to full capacity; the glottis is then closed, and the lungs are forcibly compressed by all the muscles of expiration, so that the pressure of air in the alveoli may rise to over 50 mm. of Hg (Kountz and Alexander, 1934; Rasmussen and Adams, 1942). The glottis is then opened and the strain on the ribs, the pressure on the pleura, and the pressure in the alveoli are suddenly released. In the case of the alveoli there must be a wave of pressure change passing from the bronchi out to the periphery of the lung. It is presumably this sudden release of pressure which is responsible for the sensation, usually experienced, that the lung is being distended. It is obvious, however, from the mechanism of coughing, that the lung is compressed and not in any sense distended. This building up of pressure followed by its sudden release must mean increased stress and strain on all the structures involved, especially on the alveolar walls, which are not robust and through which passes this wave of pressure change. A chronic bronchitic may cough scores of times in a day, and it seems reasonable to suppose that this type of stress and strain, or wear and tear, repeated over many years, could in time produce the loss of elasticity and degenerative changes observed in the alveolar walls. In many respects there is an analogy in hypertension, in which, without any overstretching of the blood vessels, there may be loss of elasticity and other degenerative changes, and these are ascribed by recent investigators to the chronic stress of raised pressure. Outside the lungs it is also reasonable to suppose that wear and tear might occur when cough is chronic. The pleura might suffer for the same reason as the alveoli, and the thoracic cage is also strained: one has only to place a hand over the costal cartilages while coughing to realize that the impact is considerable.

In asthma there may be the same degree of change in intra-alveolar pressure as in coughing, and although the change is not so sudden, it is repeated more often (v. Neergaard and Wirz, 1927; Hartwich, 1930). This stress and strain repeated many thousands of times a day should have the same effect as chronic cough. The mechanism of the experimental production of so-called emphysema by tracheal stenosis in animals is presumably the same (Paine, 1940a, 1940b). It is also true that during an acute attack of bronchial asthma the increased expiratory resistance may lead to distension of the lung, which tends to subside when the attack is over. This temporary distension does not rupture the alveolar walls, or in fact lead to any permanent damage, but it probably does add to the stress and strain produced by the other factors I have described.

Almost all patients with emphysema suffer from either chronic bronchitis or asthma, and in these cases the explanation of the pathogenesis of emphysema which I have given is the most likely one. It is also true that some patients with chronic bronchitis or asthma develop emphysema comparatively rapidly, while others never become emphysematous. The forcefulness of the cough is obviously an important factor, and age is another. To discuss the changes in resistance to wear and tear or to trauma that occur in senescence would in itself occupy several lectures, but no one would dispute that with age there is a decrease in the resistance of the tissues. This is particularly true of the elasticity of the blood vessels; and with advancing years there may also be diminished resistance in the lungs, and in the elastic structures of the thoracic cage, to the stress and strain of cough or of asthma, and this diminution in resistance may take place earlier in some individuals than in others.

There still remains to be explained the unusual case of emphysema with no previous history of asthma or chronic bronchitis. I have seen three such cases when shortness of breath preceded the onset of cough, and in one the diagnosis was confirmed at necropsy. I can only suggest that, very rarely, the stress and strain on the lung which occurs with ordinary breathing may be sufficient to destroy its elasticity.

Other aetiological factors have been suggested, and a hereditary tendency to emphysema is one. This may be so, but I have not seen it except indirectly as a possible familial tendency to asthma and chronic bronchitis. Hard manual labour is another, but my own belief is that this is only of importance in that it increases the incidence of chronic bronchitis and accelerates the process of ageing. It is also a striking testimony to the conservatism of medical teaching that there is scarcely a textbook of medicine which does not quote glass-blowing and the blowing of wind instruments as probable or possible causes of emphysema; and this in spite of ample evidence in the literature that no such aetiological relationship exists (Christie, 1939). The original statement of Laennec, made in 1819, based on no actual cases, has been copied from textbook to textbook over a period of 120 years.

Diagnosis

I believe it is those with little clinical experience who think they have a clear conception of the diagnosis of emphysema. The pathology of the disease as taught is quite precise and the description of its clinical manifestations is equally so, but the differential diagnosis is not even mentioned in most textbooks. It is those with wide clinical experience who have expressed difficulty in interpreting the so-called physical signs of emphysema. I have discussed the diagnosis of emphysema with many clinicians, and have found a wide divergence of opinion as to the reliable signs of this disease. All were agreed that the physical signs as traditionally described could be of value in diagnosis but could also be misleading. Obliteration of the cardiac dullness, diminished chest expansion, reduction in vital capacity, and the radiograph were each suggested as being most valuable in doubtful cases. I can find no agreement—in fact, there is wide disagreement—with regard to the relationship of the barrel-chest phenomenon to emphysema. Some believe, and teach, that the physical signs associated with the barrel-shaped chest, if definite, necessarily mean emphysema, although they admit that the lesion may neither progress nor lead to symptoms. Others believe that the barrel-chest phenomenon may occur quite independently of emphysema, and that in these circumstances it is of no significance. Such a divergence of opinion as to the diagnostic criteria naturally makes for scepticism, and attempts to correlate the clinical diagnosis of emphysema with the facts as revealed by necropsy have led to further doubts. It has been stated that only a small proportion of those diagnosed at necropsy are recognized during life, and it has also been shown that many of those with barrel-shaped chests furnish no evidence of emphysema when they come to the post-mortem table (Cabot, 1927; Davidson, 1936; Roelsen, 1938). For this reason some writers have gone so far as to say that the physical signs of emphysema are meaningless. I believe that this is certainly untrue, just as I believe that the textbooks are misleading, to say the least, in the neat array of physical signs which they present.

In order to determine the reliability of these physical signs Dr. P. Hill and I have reviewed 72 cases diagnosed on the post-mortem table as generalized hypertrophic emphysema. All of these cases came from the medical wards of the London Hospital, but an accurate and detailed case history was not always available, as some were moribund on admission. A history of chronic cough was given in 94%, and in a quarter of these it was associated with mild or severe asthma. Dyspnoea on exertion was present in 78% of the 45 case histories in which mention was made of the presence or absence of this symptom. In the majority of cases, however, the physical signs of emphysema were not observed. In 35% there was enough evidence to suggest an "emphysematous chest," but in only 13% could this evidence be said to be complete. This series of cases suggests that a more reliable guide to a diagnosis of emphysema than the physical signs is a history of chronic cough or asthma, associated with dyspnoea on exertion. I have therefore investigated another series of 25 patients who gave such a history and in whom no cause of dyspnoea other than emphysema could be discovered. In 92% of them two or more of the physical signs of emphysema were present, and in these a diagnosis of "emphysematous chest" might have been made; in 90% expiration was prolonged, but the incidence of any other individual physical sign was not more than 72%, and only 44% showed the traditional picture of emphysema. The average age of these patients was 54 years, and as a control to this series I have analysed a group of 40 patients, all over the age of 45, none of whom had a history of either cough or dyspnoea. In 9 of them the chest might have been said to be "emphysematous," and 4 presented the classical physical signs of emphysema.

The only conclusion that can be drawn from these series of cases is that the signs of emphysema are unreliable; they may be absent in patients suffering from the disease, and are not uncommonly present in patients without emphysema. Several special diagnostic methods have been recommended; but these are seldom used, and their reliability has not been established. The vital capacity in emphysema is usually diminished. I have made this measurement on 25 cases, all of them with a history of chronic bronchitis and dyspnoea on exertion. In 20 the vital capacity was less than 70% of the normal standard, as calculated from the surface area, and in 13 it was less than 60%. There is a natural tendency during senescence for the vital capacity to diminish; but at any age a vital capacity below 60% of the normal is significant, as is less than 75% of normal before the age of 50.

The diagnostic value of the vital capacity is enhanced if this measurement is made on the type of recording spirometer which is employed in the routine estimation of the basal metabolic rate. Such a record not only gives a measure of the vital capacity but can also be used to indicate the loss of elastic recoil in the emphysematous lung. I have discussed these tests in detail elsewhere (Christie, 1934; Meakins and Christie, 1934); they are based on the fact that when a normal individual takes a deep breath his lungs recoil passively to their original size because of their elasticity. In emphysema this recoil is abolished or impaired, and the lung has to be compressed by the muscles of expiration. After a full inspiration, therefore, the lung is seldom deflated to its original size when the patient is asked to expire forcibly. There are two other manifestations of loss of elasticity which can be demonstrated by this type of respiratory tracing: after a full inspiration the patient is incapable of deflating his lung as efficiently as after an inspiration of moderate depth, and the level at which the patient breathes is irregular.

During the past ten years I have performed this test on 66 patients with chronic bronchitis and dyspnoea on exertion: in 30 these characteristics were obvious and constant, in 26 the tracings were suggestive of loss of elastic recoil, and in the remaining 10 no abnormality could be detected. Of 116 controls, 11 would not co-operate, none showed definite loss of elasticity, and only 4 were suggestive of slight loss of elasticity. The main value of these respiratory tracings is that it is only patients with emphysema who consistently show loss of elastic recoil; admittedly this is true of only about 50% of cases, but most of the others give tracings which are suggestive. The drawbacks of this test are obvious. Except in hospitals where the basal metabolic rate is measured by means of a recording spirometer, special equipment is required. The test has to be performed by someone with special knowledge of respiratory tracings, patience is required to ensure that the subject knows what is wanted of him, and I do not expect that this test will be used except perhaps by those who have a particular interest in emphysema.

It is easier to measure the chest expansion than the vital capacity, but unfortunately this also diminishes with age. In my experience about 60% of patients with emphysema have a chest expansion of less than one inch, but I have seen it reduced to half an inch in a man aged 65 in whom there was no other evidence to suggest pulmonary or cardiovascular disease.

In recent years the radiograph has been suggested as a reliable guide to emphysema. This has not been my experience. In 11 cases in which the diagnosis was confirmed at necropsy, radiographs suggested emphysema in only 5. In 25 other cases, in which diagnosis was made on clinical grounds, the value of the *x*-ray film was approximately the same.

A diagnosis of emphysema based only on physical signs is therefore unreliable, since these depend largely on the barrel-chest phenomenon, which may occur independently of this disease. In my opinion the diagnosis should only be considered certain when dyspnoea on exertion, of insidious onset, not due to bronchospasm or left ventricular failure, appears in a patient who has some of the physical signs of emphysema together with chronic bronchitis or asthma. The absence either of dyspnoea or of physical signs should cast doubt on the diagnosis, but should not exclude it; the more physical signs present in a patient with chronic bronchitis or asthma, the greater is the likelihood that he has emphysema. I must confess, however, that in the absence of dyspnoea I am always uncertain as to diagnosis and prognosis, but when dyspnoea of the type described above appears in a patient with chronic bronchitis I am very seldom in doubt. A large series of cases will have to be followed up over many years before the necessary criteria for early diagnosis can be established. Until this has been done I feel that the terms "emphysematous" and "emphysematous chest" should be discarded, as it is their use which has been partly responsible for the lack of progress in the understanding of emphysema since its classical description over a century ago by Laennec. These terms are quite misleading, and should be replaced by some such expression as "barrel chest" or "barrel-chest phenomenon," reserving the diagnosis of emphysema for those patients who have the symptoms as well as the signs of the disease. I feel that if this were done the chances of progress would be increased, particularly with regard to the use of special diagnostic procedures.

Treatment

The treatment of emphysema is essentially symptomatic, as elastic tissue cannot regenerate and nothing can restore the structure of the lungs. Until heart failure supervenes, the only symptom, other than cough, is dyspnoea on exertion, and I will first emphasize the value of ephedrine. Although there may be no evidence of bronchospasm or resistance to respiration, the administration of ephedrine not infrequently relieves the dyspnoea of emphysema. A possible explanation of this effect is that the bronchioles leading to the over-distended air sacs and bullae are less capable of changes in calibre than those leading to healthier parts of the lung; bronchospasm, although not clinically manifest, would in this case increase the proportion of the inspired air deflected to these useless parts of the lung, and the relief of bronchospasm with ephedrine would improve the efficiency of ventilation and thus relieve dyspnoea.

Several procedures, the purpose of which is to deflate the lung, have been described, and it is said that these increase the efficiency of respiration. Pneumothorax is one, and relief of dyspnoea from this procedure, both in man and in horses suffering from this disease, has been claimed. More popular in this country, and less dangerous, are respiratory exercises designed to teach the patient to deflate the lung and to increase the use of the diaphragm. A well-fitting abdominal belt will also raise the diaphragm by increasing intra-abdominal pressure, and, more recently, pneumoperitoneum has been suggested for the same purpose. All these procedures have been recom-

mended, and various theories have been put forward to explain the beneficial effects observed. Two effects are common to most of them. First, the diaphragm is raised so that its convexity is increased and its efficiency therefore enhanced, and it was for this purpose that I first suggested the use of the abdominal belt. Secondly, the lung is deflated by these procedures so that it contains less air. The decrease in volume is, however, very small—so small that it can hardly be measured—and the beneficial effects are probably for the most part due to increased efficiency of the respiratory musculature. It is also possible that with greater collapse of the superficial bullae efficiency of ventilation may be increased.

When heart failure supervenes oxygen should be given, as the added insult of anoxia to a heart that is failing for other reasons greatly lessens the chances of recovery. Recovery from heart failure in emphysema is uncommon, but I have recently had a patient who recovered from two attacks, during both of which he was moribund. In the first oxygen was administered continuously for one week, and in the second for 10 days. He subsequently died during a third attack, and the diagnosis of emphysema was confirmed at necropsy.

The treatment of emphysema is thus far from being hopeless, although it is essentially symptomatic. Ephedrine should be tried in all cases. Respiratory exercises or an abdominal belt, or both, may increase considerably the tolerance to exercise. And lastly, when heart failure supervenes oxygen therapy is of the greatest value.

Summary

In this paper the disturbance of function in emphysema, and its mechanism as revealed by experiment at the bedside, are emphasized, and the bearing of these clinical observations on the natural history of the disease and its diagnosis is discussed. The primary lesion is loss of elasticity, the most common cause of which is the stress and strain of cough or respiratory obstruction on the structures concerned in respiration, the process being comparable to the loss of elasticity which occurs in blood vessels following hypertension. Almost all the symptoms and signs of emphysema, and also the over-distension of air sacs and formation of bullae, can be explained on the basis of loss of elasticity, and it is suggested that it is the wastage of ventilation on this "pathological dead space" which is responsible for the dyspnoea and the impairment of haemo-respiratory exchange which occur. The difficulties and fallacies in the diagnosis of emphysema are described, and it is suggested that the present state of confusion would be diminished if the diagnosis were reserved for those who have symptoms as well as signs of this disease.

REFERENCES

Anthony, A. (1936). *Verh. Dtsch. Ges. inn. Med.*, **48**, 244.
Beitzke, H. (1925). *Dtsch. Arch. klin. Med.*, **146**, 91.
Cabot, R. (1927). *Physical Diagnosis*, Baltimore.
Christie, R. V. (1934). *J. clin. Invest.*, **13**, 295.
—— (1936). *Quart. J. Med.*, **5**, 327.
—— (1939). *Edinb. med. J.*, **46**, 463.
—— and McIntosh, C. A. (1934). *J. clin. Invest.*, **13**, 279.
—— —— (1936). *Quart. J. Med.*, **5**, 445.
Cournand, A., and Berry, F. B. (1942). *Ann. Surg.*, **116**, 532.
—— Lassen, H. C. A., and Richards, D. W. (1937). *J. clin. Invest.*, **16**, 9.
Davidson, M. (1936). *Proc. roy. Soc. Med.*, **39**, 1315.
Hartwich, A. (1930). *Z. ges. exp. Med.*, **69**, 482.
Hoover, C. F., and Gammon, J. E. (1915). *Arch. intern. Med.*, **15**, 501.
Hurtado, A., Fray, W. W., and McCann, W. S. (1933). *J. clin. Invest.*, **12**, 833.
—— Kaltreider, N. L., and McCann, W. S. (1935). Ibid., **14**, 94.
Kountz, W. B., and Alexander, H. L. (1934). *Medicine*, **13**, 251.
Loeschke, H. (1928). *Handl. spez. path. Anat. Histol. Berlin*, **3**, 612.
Meakins, J. C., and Christie, R. V. (1934). *J. Amer. med. Ass.*, **103**, 384.
—— and Davies, H. W. (1925). *Respiratory Function in Disease*, Edinburgh.
v. Neergaard, K., and Wirz, K. (1927). *Z. klin. Med.*, **105**, 51.
Paine, J. R. (1940a). *J. thorac. Surg.*, **9**, 550.
—— (1940b). Ibid., **10**, 150.
Peters, J. P., and van Slyke, D. D. (1931). *Quantitative Clinical Chemistry*, **2**, Baltimore.
Rasmussen, R. A., and Adams, W. E. (1942). *Arch. intern. Med.*, **70**, 379.
Roelsen, E. (1938). *Acta med. scand.*, **95**, 452.
Tendeloo, N. Ph. (1925). *Allgemeine Pathologie*, J. Springer, Berlin.
Thies, O. (1932). *Virchows Arch.*, **284**, 796.

The Trustees of the Nuffield Provincial Hospitals Trust have set aside a substantial sum for grants towards the establishment of preliminary training schools for nurses for the benefit of groups of hospitals, both voluntary and municipal. Schemes will be considered from Regional and Divisional Hospitals Councils or other joint hospital bodies recognized by the Trust. For areas in which these bodies do not exist, the Trustees have agreed to receive schemes through the appropriate area committee of the British Hospitals Association, but they must cover facilities for local authority as well as voluntary hospitals.

INFANT FEEDING IN RELATION TO MORTALITY IN THE CITY OF BELFAST

BY

JAMES DEENY, M.D., M.Sc., F.R.C.P.I.

AND

ERIC T. MURDOCK, B.Sc., Ph.D., A.I.C.

(Lurgan)

In the course of a recent study of the infant mortality problem in Belfast we have collected some facts relating to infant feeding. For this city the death rate of children under 1 year has been high, and during the past four years has ranged from 85 to 122 per thousand live births. In the survey data were obtained concerning 554 deaths, or 84% of the total deaths of legitimate infants occurring from June, 1941, to June, 1942, in Belfast (completed by Dec., 1942). The total legitimate births in Belfast for this period were 7,778, 3,359 occurring in the first six months. Material was also collected from a group of children who had survived 1 year of life in the city. This control sample was established by selecting every fifth child born during the first six months of the period, and, allowing for wastage, finally numbered 477.

In order to maintain constant values the information was obtained by one investigator (E. T. M.) from a personal visit to the parent or guardian. Parents usually possess vivid memories of the circumstances of the death of their children, and were generally willing to discuss the matter. Cases were excluded when the fatal illness was due to hardships resulting from previous air raids, and when families had moved from the city and could not be followed up. Information was recorded concerning the size of the family (and previous infant deaths), income, housing, the care and feeding of the child, domestic hygiene, the ante-natal and obstetric attention, cause of death, the medical, nursing, hospital, and other factors relating to the fatal illness—entailing more than 40 questions in each case. In the control group somewhat similar data were obtained. This paper deals only with the information we have secured concerning infant feeding.

Infant Feeding in Belfast

Feeding is an important factor associated with infant mortality, and in any examination of the problem a knowledge of the general character of the infant feeding practised is necessary. This subject was considered under three main headings—viz.: (1) the principal foods given to ordinary children for the first six months of life; (2) the food given for the second six months; and (3) foods the infants received before the fatal illness. It is obvious that this method of inquiry does not yield results in great detail, but it will present a fairly accurate picture of the main methods of infant feeding used in the city.

TABLE I.—*Analysis of Feeding of Infants in the Control Group, during the First Year of Life*

Method of Feeding	Aged 0–6 months	Aged 6–12 months
Breast-fed (with or without "supplement")	337 (71%)	157 (33%)
Cow's milk	61 (13%)	33 (7%)
Dried proprietary milk	40 (8%)	19 (4%)
Cereal foods	39 (8%)	58 (12%)
Cereal and "mixed feeding"	—	68 (14%)
"Mixed feeding" and cow's milk	—	142 (30%)
Total	477 (100%)	477 (100%)

For the children in the control group, which is representative of living children in Belfast, we found (see Table I):

1. *During the first six months* almost three-quarters received breast milk, with or without a "supplement," as the principal food. Of the remainder, a slightly larger proportion were fed on cow's milk than on cereals or dried proprietary milk.

2. *During the second six months* the same children showed a change in diet, with a decrease in breast-feeding. Only one-

third received breast milk, another third were given "mixed feeding" and cow's milk, and the remainder were fed on such diets as cow's milk alone, dried proprietary milk, or cereal foods with or without "mixed feeding."

3. *The major feeding before the fatal illness* was studied in the mortality group, as we thought that this would give most information on the relation of feeding to mortality. From this, and by relating the figures obtained to the age at death (Table II), it was found that the feeding followed the general

TABLE II.—*The Major Feeding before the Fatal Illness, Classified according to Age at Death*

Feeding before Fatal Illness	Age at Death						Total
	Under 7 days	1–4 wks.	4 wks.–2 mths.	2–3 mths.	3–6 mths.	6–12 mths.	
Breast-fed (with or without "supplement") ..	2	27	29	21	23	24	126
Cow's milk	—	5	20	23	47	36	131
Dried proprietary milk ..	1	7	6	9	26	25	74
Cereal foods	—	2	—	4	12	20	38
Other methods	—	—	1	—	—	1	2
Total	3	41	56	57	108	106	371
Congenital or premature cases not affected by feeding	133	44	6	—	—	—	183
Total	136	85	62	57	108	106	554

trends experienced in such groups—i.e., the amount of breast-feeding diminished as age increased, with a correspondingly greater use of the other foodstuffs. Age of death seemed to be the major factor influencing the manner of feeding before the fatal illness.

Incidence of Breast-feeding

Nowadays so many babies receive supplements that it was thought advisable to use a broader definition of breast-feeding. Consequently, in the following consideration of the duration of breast-feeding the infant was classified as breast-fed if the major part of its diet was breast milk.

It is apparent that there is a substantial difference in the extent of breast-feeding between the mortality and control groups (Tables I and II). These findings cannot be directly compared, as the same aspects of feeding were not considered in the two groups. However, figures giving a simple comparison can be obtained from our data by determining the percentage of babies breast-fed at various ages. In the mortality group the percentage breast-fed was based on the number of infants alive at these ages, and was found by relating the duration of breast-feeding to the age at death as in Table III. This table also gives the duration of breast-

TABLE III.—*Number of Infants who Died classified according to Duration of Breast-feeding and Age at Death, and Number of Infants who Survived (Control Group) classified by Duration of Breast-feeding*

	Mortality Group (Dead Children)								Control Group (Living Children)
	Age at Death							Total Deaths	
	Under 1 day	1–7 days	1–4 wks.	4 wks.–2 mths.	2–3 mths.	3–6 mths.	6–12 mths.		
Unsuccessful attempt to establish breast-feeding	—	9	6	1	—	5	4	25	14
Died before breast-feeding could be established	67	27	—	—	—	—	—	94	—
Not breast-fed	—	19	40	21	23	22	18	143	30
Breast-fed for:									
Less than 1 month	—	14	39	11	9	26	22	121	74
1 mth. but less than 2 mths.	—	—	—	29	7	17	7	70	35
2 " " 3 "	—	—	—	—	18	11	3	32	35
3 " " 6 "	—	—	—	—	—	27	18	45	83
6 " " 9 "	—	—	—	—	—	—	} 24	24	50
9 months and over	—	—	—	—	—	—			156
Total	67	69	85	62	57	108	106	554	477

feeding for the control group. At 1 month of age three-quarters of the children in the control group were breast-fed. Of the infants in the mortality group, at 1 month only one-half were breast-fed—a striking contrast. The proportion breast-fed is considerably less at each month of age in the mortality group than in the control group (Table IV). Despite the smallness of the numbers in the sample this conclusion is decisive.

TABLE IV.—*Comparative Incidence of Breast-feeding in Infants who Died and in those who Lived*

Age of Child in Months	Numbers Living		Numbers Breast-fed		Percentages Breast-fed	
	Mortality Group	Control Group	"M" Group	"C" Group	"M" Group	"C" Group
1	333	477	171	359	51	75
2	271	477	101	324	37	68
3	214	477	70	289	33	61
6	106	477	24	206	23	43
9	—	477	—	156	—	33

Duration of the fatal illness of infants in the mortality group does not account for a significant part of the difference between the mortality and control percentages of breast-feeding at each age; a special analysis shows that of the 333 in the mortality group who survived one month, 60—of whom 40% were breast-fed—were sick at this age, leaving 273 who were still healthy. Of the latter, the number breast-fed was 147, or 54% of the total not sick. Thus the omission of sick children from the mortality group only increases the percentage of breast-fed babies from 51 to 54, and this is still very significantly different from the 75% of breast-fed observed in the controls.

Social Factors and Incidence of Breast-feeding

The possible influence of some social factors as a cause of the marked disparity between the incidence of breast-feeding in the mortality and that in the control group was studied: income, housing, and domestic hygiene were considered.

Income in relation to breast-feeding was analysed by dividing the infants of both groups into three different income grades. These were based on the amount, less rent, available to the mother for household purposes per head (including the child in question) per week. In the mortality group the amount reckoned was the weekly average for the month preceding the fatal illness. A similar average when the children were 6 months old was taken in the control group. The proportion of infants breast-fed in each income level, at various ages, was determined for the mortality and control groups, and the results show that the variations in income considered did not affect the measure of breast-feeding in either group.

Housing.—In ascertaining the effect of housing, the infants of the two groups were arranged in classes according to the number of persons per room in each house, and the proportion of children breast-fed in each class found.

Domestic Hygiene.—The influence of domestic hygiene was considered by dividing the homes of the infants into three grades: (A) very clean and showing maternal efficiency; (C) dirty or showing maternal inefficiency; (B) intermediate conditions. These analyses (Table V) show plainly that changing values in housing or domestic hygiene did not appreciably influence breast-feeding.

Order of Birth and Other Factors

Order of Birth.—This has an important effect on infant mortality, and on this account the possibility of a relationship between birth order and breast-feeding was examined. The infants in the mortality and control groups were classed according to this factor, and the incidence of breast-feeding determined in the classes for the various ages. While the figures obtained (Table V) may indicate vague trends relating breast-feeding incidence and birth order, statistical treatment shows that these tendencies are not significant for the numbers involved.

Cause of Death.—The incidence of breast-feeding was considered in respect of the following three causes: (*a*) gastro-enteritis; (*b*) respiratory infections; and (*c*) other causes. "Other causes" include prematurity and injury at birth—from the latter group only eight babies dying survived one month. The slightly lower incidence of breast-feeding among infants dying from gastro-enteritis (Table V) is not statistically significant, and could be ascribed to chance variation.

Occupation.—As very few of the mothers were engaged in industry this factor was neglected. Any attempt to establish vocational grades based on the occupation of the father failed owing to the number serving in the Forces.

The age of the mother was not sought. It was considered that the numbers in the samples would not be sufficient to give a satisfactory analysis for the necessary age groups. (See Campbell, 1929; Burns, 1942.)

Statistical Note

In Table V the figures relating the factors to the incidence of breast-feeding at 1 month and at 2 months of age are given. Results at 3 and 6 months furnish the same conclusions, but are not included in the table. A number of "sorts" for each social factor were involved in calculating these figures. The standards of the social factor were related (*a*) to the age at which breast-feeding ceased, and (*b*) to the age at death. From analysis *a* the number of children in the standard classes of the social factor at each month of age was determined, and analysis *b* gave the total number of children alive in the standard classes at these ages for the mortality group.

The significance of the figures in this table was tested by a χ^2 analysis. All differences from the expected figures for a "no relationship" consideration may be ascribed to chance. For each χ^2 analysis, P is greater than 0.3—except in the case of birth order in the control group, when, with $\chi^2 = 11.8$ and $n = 8$, P is between 0.2 and 0.1.

TABLE V.—*Incidence of Breast-feeding at Ages of 1 Month and 2 Months related to Various Factors*

		1 Month				2 Months			
		Numbers Breast-fed		Percentages Breast-fed		Numbers Breast-fed		Percentages Breast-fed	
		Mortality Group	Control Group	"M" Group	"C" Group	"M" Group	"C" Group	"M" Group	"C" Group
(i) Income less rent per head per week	Under 10s.	75	85	51	77	51	74	43	67
	10s. and under 15s.	52	118	55	75	27	114	35	72
	15s. and over	44	156	48	75	23	136	31	65
(ii) Housing: Number of persons per room (including the child in question)	Less than 1	30	96	62	75	14	85	37	66
	1 but less than 1½	57	158	47	77	34	142	33	69
	1½ and including 2	38	67	49	77	24	63	39	72
	More than 2	46	38	53	68	29	34	43	61
(iii) Domestic hygiene	Condition A	56	165	47	76	29	150	31	69
	Condition B	86	157	55	75	51	140	41	67
	Condition C	29	37	48	71	21	34	40	65
(iv) Order of birth	First births	55	133	56	73	33	118	43	64
	Second births	28	69	49	71	14	61	29	63
	Third births	30	51	48	82	16	46	30	74
	Fourth and fifth births	22	58	46	82	13	55	33	77
	Sixth births and others	36	48	54	75	25	44	45	69
(v) Cause of death	Diarrhoea and enteritis	44	—	48	—	25	—	33	—
	Respiratory infections	63	—	55	—	41	—	39	—
	Other causes	63	—	52	—	35	—	38	—

Comment

In the first part of this paper it has been shown that three-quarters of the children who had survived one year in Belfast were principally breast-fed, with or without a supplement, during the first six months of life. Of the small numbers of these children not breast-fed less than one-third were fed on cereal foods—the remainder received dried proprietary milk or cow's milk. For the larger part of the second six months one-third were breast-fed. This analysis of the feeding methods employed takes into account only the nature of that feeding. Such important considerations as the quality, quantity, or nutritive value of the foods could not be examined. Assessing the feeding practised in the city by the prevalence of breast-feeding or by the nature of the artificial foods given, the infant feeding in general use can be considered as adequate. This conclusion is supported by the finding, previously reported, that the incidence of gastro-intestinal and respiratory infections in the control group is low, although these two diseases are the cause of 42% of the deaths in the mortality group (Deeny and Murdock, 1943).

Even if the wider definition of breast-feeding employed in this study is taken into account the figures obtained for the control group show an incidence of breast-feeding in Belfast comparable to that of similar groups in other cities with a lower infant mortality rate. In addition a greater proportion of infants are breast-fed for a longer time (Blessing, 1937; Spence, 1938; Robinson, 1939; Hughes, 1942).

The study reveals a marked difference in the incidence of breast-feeding between the infants who die and those who live. Of the former 51% were breast-fed at one month, while 75% of the latter were breast-fed at that age. This result is in agreement with the finding of Grulee *et al.* (1934) that the infant mortality rate is much higher in artificially fed children.

Our results show that the social factors of income, housing, and domestic hygiene have no significant influence in determining the extent of breast-feeding in either the mortality or the control group. This endorses the biological nature of the process of breast-feeding. The investigation of the effect of order of birth or cause of death on breast-feeding revealed no appreciable relation. The consistency of these findings is noteworthy. No statistical relationship could be demonstrated throughout the subdivisions of each factor considered, either in the mortality or in the control group.

From the data it has not been possible to determine any further relation between the incidence of breast-feeding and mortality than that shown. However, it is interesting to consider the significance of this lowered incidence of breast-feeding in the mortality group. Would an increase of breast-feeding in this group reduce the mortality rate appreciably? We suggest that it would not, and that this lowered incidence of breast-feeding should be regarded more as an effect than as a cause of unsuccessful reproduction.

Summary

Facts concerning infant feeding obtained in a study of 554 deaths (84% of the total) occurring in one year in Belfast, and in a control group of 477 living infants, are presented.

The extent of breast-feeding in the infants who died was much less than in the control group.

The factors of income, housing, domestic hygiene, order of birth, and cause of death are shown to have no appreciable influence on the extent of breast-feeding in Belfast.

A statistical treatment of the results is given.

This work is a portion of a study of the infant mortality problem in Belfast which is being presented to the Statistical and Social Inquiry Society of Ireland. We wish to thank the Medical Research Council of Ireland for its generous assistance. We are grateful to the Director, the Senior Statistician, and Staff of the Statistical Branch of the Department of Industry and Commerce, Eire, for machine compilation of the data and technical advice, and appreciate their courtesy and kindness.

REFERENCES

Blessing, R. (1937). *J. Pediat.*, **10**, 792.
Burns, C. M. (1942). "Infant and Maternal Mortality," University of Durham.
Campbell, J. (1929). Ministry of Health Reports on Public Health and Medical Subjects, No. 55, London.
Deeny, J., and Murdock, E. T. (1943). *Ulster med. J.*, **12**, 117.
Grulee, C. G., Sanford, H. N., and Herron, P. H. (1934). *J. Amer. med. Ass.*, **103**, 735.
Hughes, E. L. (1942). *British Medical Journal*, **2**, 69.
McNeil, C. (1942). Ibid., **2**, 271.
Robinson, M. (1939). *Arch. Dis. Childh.*, **14**, 259.
Spence, J. C. (1938). *British Medical Journal*, **2**, 729.

AN UNUSUAL CASE OF CEREBRAL MALARIA

BY

CHARLES S. D. DON, M.D., M.R.C.P.

Temp. Major, R.A.M.C.; Medical Specialist

AND

P. F. MEYER, M.D.

Captain, R.A.M.C.; Graded Physician

This case is reported because of the large number of parasites in the blood film and the severe clinical signs, in spite of which recovery followed.

Clinical History

The patient, a soldier aged 22, was admitted to a C.C.S. in North Africa on July 10, 1943. We subsequently learnt that while on local leave in North Africa he became ill, but attributed the illness to the heat and did not consult a medical officer. He had no clear memory of how he reached this C.C.S.; he last remembers travelling in a train with other soldiers, who finally took him to the central M.I. room of this area.

He was admitted late at night, about five or six days after the onset of the illness. On examination he complained of headache and vomiting. He was drowsy but not unconscious, and could answer questions reasonably well. The temperature was 102.4° F. The spleen was easily palpable. The urine was of normal colour. Blood slides showed a very heavy malarial infection. The thick film was crowded with parasites. Examination of the central parts and the edges of the thin film revealed that, on an average, 35% of the red cells contained parasites. Of the infected cells 82% had one ring, 17% had two rings, and 1% had three or four rings per cell. Several crescents of *Plasmodium falciparum* were also seen.

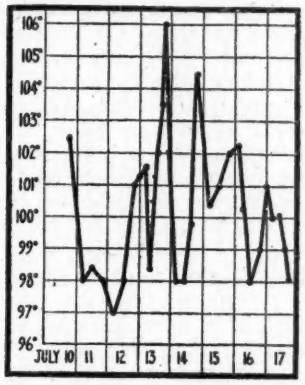
He was given 10 gr. of quinine dihydrochloride in 20 c.cm. of water intravenously and 12 gr. by mouth at 11.30 p.m., the latter dose being repeated in six hours. Next day his condition was only fairly satisfactory; he was still drowsy, but he took 10 gr. of quinine six-hourly by mouth without vomiting. The temperature remained normal all day. A blood film showed large numbers of parasites to be still present. He took fluids fairly well, but passed only very small amounts of urine.

Early on the morning of July 12 the patient was found to be quite unconscious, cold, and collapsed. The tongue was hard and dry. The temperature was 97° F., the blood pressure 80/60, and the pulse 90 per minute. Hiccup was present, and breathing was shallow. As the patient appeared to be dying he was given an immediate injection of nikethamide (coramine). No urine had been passed since the previous night and the bladder appeared to be empty, but when a catheter was passed an ounce of urine was obtained which contained albumin but no blood. Microscopically, granular and hyaline casts were seen. A glucose-saline drip was put up, and by this route he was given in the course of the day 30 gr. of quinine dihydrochloride and two blood transfusions, each of 500 c.cm., without a reaction of any kind. The patient was Group B, and each donor was of this group. The serum and red cells of the patient and of each donor were cross-matched and the results read microscopically at the end of one hour. A blood film of the patient showed large numbers of parasites to be still present. The haemoglobin before the transfusion was 70% (Sahli). By evening his condition had improved; the B.P. had risen to 105/80, and the temperature was 101° F. No urine was passed that day.

The following morning—July 13—the patient was semiconscious, and was able to take fluids by the mouth. The temperature was normal, the conjunctivae showed slight jaundice, and a specimen of urine voluntarily passed was typical of that seen in blackwater fever. Spectroscopic examination revealed the presence of oxyhaemoglobin. No red cells were seen in the centrifugalized deposit, but granular and hyaline casts were present. While the compatibility of the transfused blood appears to have been established by indirect grouping and careful direct cross-matching, the possibility remains that the haemolysis observed was not due to quinine but to antibodies in the recipient's blood which could not be detected by the methods at our disposal. There was no history of any previous blood transfusion. The intravenous drip was continued; no further quinine was prescribed, and the patient was given by mouth one drachm of sodium bicarbonate hourly to alkalinize the urine rapidly. In the course of the day he was given two intramuscular injections of mepacrine methanesulphonate (atebrin musonate), each of 0.1 g., and the urine gradually returned to a normal colour. At 6 p.m. the temperature was 103.6° F.; he was given mepacrine hydrochloride (atebrin) 0.1 g. by mouth, and this was repeated in 6 hours. At 9 p.m. the temperature, which had been taken hourly, had risen to 106° F., and the patient, after having remained semiconscious all day, became delirious and restless. Cold sponges were given half-hourly until 11 p.m., when the temperature had fallen to 102° F. The intravenous drip was stopped, as oedema of the sacrum was found to be present; he was passing urine freely.

On July 14 the patient's general condition was much improved; for the first time he was quite conscious and rational. The morning temperature was 98° F. A blood film showed no parasites; the red cells exhibited a marked degree of macrocytosis, and megaloblasts were present. The urine still contained albumin and casts. Mepacrine hydrochloride was continued in a dosage of 0.1 g. t.d.s. That evening the temperature rose to 104.8° F.

On July 15 his general condition was better, but he was found to have thrombophlebitis of the left axillary vein (this arm was used for the drip) with oedema of the forearm and hand. The morning temperature, which so far had always been normal, was 100.4° F.



Temperature chart of the case.

There were rales over the lower lobes of both lungs but no dullness. It was decided that he had a hypostatic pneumonia, and was put on sulphadiazine 1 g. four-hourly. The temperature reached 102° and 101° F. on the next two nights, and thereafter settled, remaining normal all day on July 21. By this time the lung signs had disappeared.

Summary

A case of cerebral malaria is reported in which 35% of the red cells were infected by one or more parasites of *Plasmodium falciparum*.

The most-pronounced cerebral symptoms came on after the patient had been on large doses of quinine for 36 hours.

Transient haemoglobinuria followed 30 gr. of intravenous quinine and two blood transfusions.

Hyperpyrexia was found to be developing after three days of intensive quinine treatment.

Hypostatic pneumonia complicated recovery.

We wish to thank Major-Gen. E. M. Cowell, the D.M.S., North African Force, for permission to submit this report for publication.

EFFECTS ON RATS OF PROLONGED FEEDING WITH THE STAPLE AFRICAN DIET

BY

JOSEPH GILLMAN, D.Sc., M.B.

(From the Department of Anatomy, University of the Witwatersrand, Johannesburg)

It is known from numerous surveys and observations that pulmonary tuberculosis and other diseases of the respiratory tract are especially prevalent among the Africans (South African negroes). It is also common knowledge that cirrhosis of the liver and especially primary cancer of the liver are very often encountered in this people. In an extensive investigation into the incidence of cancer in South Africa, Berman (1940, 1941) has shown that in a series of 253 cases of cancer of all organs in adult male Africans working in the Witwatersrand gold mines no fewer than 222, or 90%, were primary in the liver. This remarkable state of affairs is even more strongly emphasized when it is remembered that liver cancer is the rarest neoplasm in Europeans.

Several factors are regarded as contributory to this high incidence of cancer, one of which is cirrhosis, which is almost invariably associated with liver cancer. There is a considerable body of opinion which regards this susceptibility to liver cancer

in the pigmented peoples as due to some racial factor. Des Ligneris (1940) has shown that extracts from the cancerous and non-cancerous livers of Africans are capable of producing neoplasms of the skin when painted on the backs of mice. The extracts from normal Europeans are said to be less potent in this respect. A similar experience has been reported by Hieger (1940).

Effect of African Diet on Rats

Malnutrition among the Africans is widespread in South Africa. The overwhelming majority feed on a diet consisting largely of maize meal (mealie pap) and sour milk. This forms the staple from the time of weaning throughout life. Before invoking a racial or a genetic factor in modifying the incidence of disease it seemed highly desirable to ascertain the effect of the African diet on normal animals. Accordingly 125 rats weighing from 40 to 50 g. were fed exclusively on mealie-meal porridge and sour milk. The mealie-meal porridge was concentrated to form a solid mass, while the sour milk was prepared from fresh milk allowed to ferment in a warm room. The animals received liberal amounts of this food. Apart from thinning of the hair over the abdomen and hind legs and roughening of the tail, no obvious signs of acute avitaminosis were recognized.

After 14 months 12 rats were killed and a full post-mortem examination was performed. The others are being studied biochemically. In all of those killed some degree of liver pathology was evident macroscopically. This varied from diffuse enlargement of the whole liver with obvious fatty change to a widespread nodular cirrhosis, the latter usually involving the left side of the liver. In one case there was complete atrophy of the left lobe, with enormous compensatory hypertrophy of the right lobe—so much so that the enlarged lobe extended almost into the pelvis. In this instance the remains of the left lobe could be seen as two small appendages of cirrhotic tissue.

In this series of rats it seemed that the liver first enlarged and underwent fatty changes and later cirrhosis supervened, or else complete atrophy of the affected lobes occurred, with corresponding enlargement of the right side. The heart was grossly enlarged, and in 9 cases one or both lungs showed recent or old-standing inflammatory lesions with areas of bronchiectasis or abscess formation. The fat in the mesentery, retroperitoneal, or subcutaneous regions was abundant. The skull was very much thickened, especially in the occipital region. The suture lines could be readily identified by the zones of great vascularity. The upper incisor teeth were particularly affected; many of them were broken, some fused, and others loose. The pituitary was enlarged, the thyroids were atrophied, and the suprarenals were small and of greyish colour. In four cases the lymph glands of the peri-oesophageal region and above the lesser curvature of the stomach or above the abdominal aorta were of dark-brown colour, due to the presence of haemosiderin. The jejunum in those cases of advanced cirrhosis contained a dark viscid material which gave the reaction of blood. It is noteworthy that although many thousands of rats were examined post mortem over a number of years no lesions in the liver of the kind described above were ever observed.

Comment

The significant features of this feeding experiment in rats are the gross pathology in the liver, the frequency of lung lesions, the enlarged heart, the abundance of adipose tissue, the thickened skull, and the dental affections, all of which occurred without any recognizable manifestations of acute vitamin deficiency.

The production of liver damage by means of a diet which forms the staple of the overwhelming number of Africans in South Africa is not without its sociological implications. It may also have repercussions in other parts of the world where economically impoverished individuals are forced to live on a diet similar to that of the African in the Union of South Africa.

This investigation was carried out in the Department of Anatomy, and all facilities were generously provided by the University of the Witwatersrand, Johannesburg. This and subsequent aspects of the problem represent, and will represent, the collaborative efforts especially of Dr. Leon Golberg of the South African Institute for Medical Research, and of Christine Gilbert, Dr. Theodore Gillman, Joyce de Bruijne, Marianne Cassirer, and Joel Mandelstam of the Department of Anatomy. The work is proceeding actively to discover the factors responsible for the lesions produced experimentally in the rat.

REFERENCES

Berman, C. (1940). *S. Afr. J. med. Sci.*, **5**, 54.
—— (1941). Ibid., **6**, 145.
Des Ligneris, M. J. A. (1940). *Amer. J. Cancer*, **39**, 489.
Hieger, I. (1940). Ibid., **39**, 496.

Medical Memoranda

Periarteritis Nodosa diagnosed Clinically

Following on the annotation on periarteritis nodosa published in the *British Medical Journal* of May 15, 1943 (p. 608), this case record may be of interest.

CASE HISTORY

A woman aged 34 was admitted to the Meath Hospital under my care in June, 1942, complaining of "asthma" for the past week. Previous to this attack she had never suffered from asthma or bronchitis or from any other serious illness. On admission her temperature was normal and her pulse rate 118. There was evidence of acute bronchitis, but otherwise physical examination revealed nothing abnormal. A chest radiograph was negative. The urine contained some albumin on admission, but no casts at any time. Investigation for protein sensitivity to foods, flowers, and animal emanations was negative. During her stay in hospital her temperature was occasionally slightly elevated, and her pulse rate after the first week settled down to 80. After a period of five weeks in hospital, during which time she was treated on general lines, the patient went home feeling fairly well.

In Sept., 1942, she began to have further attacks of asthma, for which she was treated with a stock vaccine by her general practitioner, who stated, however, that even a small dose of the vaccine often gave her quite a severe general reaction; and finally the treatment had to be abandoned. Shortly after this she got a thrombosis of the right long saphenous vein, which gave rise to fever at night-time. She recovered completely from the thrombosis, but in Nov., 1942, started to complain of extreme general weakness and malaise and a raised evening temperature. A blood culture done at this time was reported to contain *B. proteus*, but this was considered to be a skin infection. Owing to the presence of petechial haemorrhages on the chest and back, a blood examination was also carried out, with the following results: R.B.C., 4,100,000 per c.mm.; W.B.C., 26,000 per c.mm.; Hb, 65%; colour index, 0.8; platelets, 110,000 per c.mm. Differential count: polymorphonuclear leucocytes 85%, lymphocytes 12%, large mononuclears 3%. Prothrombin clotting time, 35 seconds.

The patient was readmitted to hospital with a temperature of 99° and a pulse rate of 96. On examination this time, in addition to her chest condition she had a very loud grating to-and-fro pericardial friction rub, heard all over the praecordium, but the heart sounds underneath did not appear to be altered. There was abundant albumin in her urine, and a catheter specimen revealed pus, red cells, and granular and hyaline casts. Her blood urea was 36 mg. per 100 c.cm. She was put on a nephritic diet. During the first week her temperature rose until it touched 103°, and her pulse rate became elevated to 120. At this stage she was treated with sulphathiazole (0.5 g. q.q.h.), but this gave rise to severe vomiting within the first 24 hours, and was discontinued. A second blood culture done a week after admission grew a staphylococcus—possibly also a skin infection—but the patient was started immediately on large doses of sulphadiazine, and during the next three weeks she received two courses each consisting of 25 g. Repeated white cell counts were done to check sulphonamide treatment; in the course of these a leucocytosis of 42,000 per c.mm. was recorded, with an eosinophilia of 9%. A fortnight or so after admission all signs of pericarditis had gradually disappeared. During her second period in hospital the patient had several attacks of asthma. At times also transitory painful lumps of about the size of a grain of rice appeared on her scalp and eyelids, and remained for about three days. In the middle of December she developed an angioneurotic oedema of the right arm. A little later there was a definite ulnar neuritis of this arm. Throughout her stay her urine was loaded with albumin, and casts were always present—granular, hyaline, and fatty. Her blood urea at all times remained within normal limits. She was discharged at the end of February at her own request.

Shortly after her return home she developed oedema of the legs, which gradually became a generalized oedema associated with ascites. During this time her urine on boiling became almost solid with albumin. In April, 1943, she had painful subcuticular lumps of about the size of a rice grain on the anterior surface of her forearms. Bullae then developed around each lump, and after from four to five days these burst, leaving punched-out crater-like ulcers with bluish-purple margins. At the same time the fingers, especially those corresponding to the ulnar distribution of the right hand, became blue, and dry gangrene ensued. The toes also became blue, but to a less degree. The patient's condition thereafter deteriorated rapidly until, about three weeks before death, the ulcers had coalesced to

form deep gangrenous excavations between the bones of the forearm, in which muscular aponeuroses could be seen. She required a great deal of omnopon during the terminal stage of her illness to combat pain. On May 18, 1943, she died.

A post-mortem examination was unfortunately out of the question, and pathological confirmation is therefore lacking, but from the clinical course of the disease I think there is little doubt as to its true nature. Alternative diagnoses of rheumatic pericarditis, acute nephritis, septicaemia, and subacute bacterial endocarditis were all considered in turn as various signs arose, but none appeared entirely to satisfy the symptomatology. The appearance of nodules and the extensive patches of gangrene on the arms, together with the obvious signs of a vascular lesion of the extremities, suggested the true diagnosis.

I am grateful to many for their help in this case, but particularly to Dr. W. O'Donnell, Prof. W. J. E. Jessop, and the late Prof. W. Boxwell, as well as to my house-physicians.

Dublin. CYRIL MURPHY, M.D., F.R.C.P.I.

Tetanus following Appendicectomy

The following case seems to be worth reporting because it developed after an operation and was successfully treated without opening up the wound.

The patient was a well-built man aged 29, employed in the N.F.S. For two weeks before admission to hospital he had been complaining of pain and paraesthesia in the finger-tips of the right hand. A diagnosis of brachial neuritis had been made by a consultant physician.

He was admitted to hospital on June 6, 1943, with a typical history of acute appendicitis of 12 hours' duration. Operation was immediately performed, and an acutely inflamed appendix was revealed. The appendix was unobstructed and not perforated, and there was but little local peritonitis. A routine appendicectomy was performed without any unusual soiling of the peritoneum or abdominal wall with the contents of the appendix, the abdomen being closed without drainage. The operation was followed by an uneventful recovery until the ninth day, except for slight pain in the hand due to the brachial neuritis. On that day the patient complained of an aching pain in the lumbar region. Examination revealed tenderness of the lumbar muscles, but no spasm. Further questioning disclosed that the ache had been present in a mild form on the sixth day. A diagnosis of lumbago was made.

On the tenth day after operation there was slight stiffness of the jaw, and examination of the lumbar muscles showed a slight but definite spasm. A tentative diagnosis of tetanus was made, and this was substantiated later in the day by the development of difficulty in swallowing. The patient also complained of the sensation of being periodically kicked in the back. This was presumably due to the onset of localized tetanic spasms. 180,000 i.u. of A.T.S. was immediately given intravenously, and chloral and potassium bromide, 20 gr. of each, were administered four-hourly.

The patient's condition deteriorated for the next two days, and he complained of tightness of the chest. Marked opisthotonos, abdominal rigidity, and trismus developed, but the bromide and chloral sufficed to keep the spasms local and infrequent. On the third day however, paraldehyde, 4 dr. twice a day per rectum, was required to control the spasms. Later the paraldehyde had to be given six-hourly in doses of 6 dr. to produce the required effect. Tribromethol was not used because of the increased liability to respiratory complications, and pethidine and chloretone were found to be less efficacious than paraldehyde.

The fifth day, when several generalized spasms were experienced, was probably the worst day. Then improvement began with disappearance of the rigidity of the abdominal wall. On the seventh day 100,000 i.u. of A.T.S. was given. As the condition improved less-sedative drugs were needed. Nutrition was maintained at a fairly adequate level throughout with glucose and milky fluids, without having to resort to a stomach tube. It was very important to feed the patient rapidly and efficiently just before the next dose of sedative was due. Then he was just conscious enough to feed, and yet the trismus was reduced and swallowing did not provoke the onset of generalized spasms.

A bacteriological diagnosis of tetanus was not made. There was a slight purulent discharge from the wound, swabs from which revealed no evidence of tetanus bacilli. Their absence was probably due to the fact that a specimen could be obtained only from the superficial part of the wound. The wound was not explored.

There was no history of any injury, and the appendicectomy wound was the only possible focus for the growth of the tetanus bacilli. The catgut used at the operation was made by a reputable firm of manufacturers. When the tetanus became manifest it was not possible to trace the batch of catgut which was used and so test it for the presence of spores. Present-day catgut is an unlikely source of tetanus spores. It seems likely that the wound was contaminated by spores from the bowel lumen. It is stated by many authorities that tetanus spores are occasionally found in the lumen of the human bowel.

I wish to thank Mr. A. H. Southam for permission to publish this case and for his helpful criticism.

A. JOLLEYS, M.B., CH.B.,
Surgical Registrar to Out-patients, Manchester Royal Infirmary.

Reviews

MENTAL GROWTH OF CHILDREN

The Mental Growth of Children from 2 to 14 Years. A Study of the Predictive Value of the Minnesota Preschool Scales. By Florence L. Goodenough and Katharine M. Maurer. (Pp. 130. $2.50 or 15s. 6d.) Minneapolis: University of Minnesota Press; London: Oxford University Press.

This is No. 19 of the series of monographs from the Institute of Child Welfare of the University of Minnesota. In it the results of repeated tests of the intelligence of the same children over a substantial period of time are described. The tests used are the Minnesota pre-school scales, revisions of the Stanford Binet and the Arthur performance scale, and the Minnesota College ability tests. At all ages the tests proved of predictive value. It was found that there is a good deal of inconstancy in the results of tests, especially in the early years, so that single tests are apt to be misleading and the longitudinal method here advocated would seem to be much more reliable. It proved difficult to follow many children through the whole 12-year period, but others of corresponding status were brought into the series, and the authors consider that their results are statistically valid.

Details of method and of results are given, and it would appear that individual mental growth does not always proceed at a fixed and uniform rate, though in the majority of cases constancy rather than inconstancy is the rule. Irregularity is most noticeable at early ages; there may be early precocity which does not last or early slowness with subsequent acceleration. Such irregularities are not necessarily due to external causes only. The old pre-school tests have not proved to be reliable, and new tests have been and still need to be worked out. The present study has shown that trustworthy indications of special abilities and differences of mental patterns can be detected at a much earlier age than was supposed. There is a suggestion that there may be a sex difference in the age at which mental tests begin to have a predictive value for future intellectual standing. It would appear that there is an initial feminine precocity which may be reversed later on. Further work is required and will be undertaken to amplify and confirm the findings.

This is a well-documented book with a useful bibliography at the end, and it will prove interesting and valuable to all who are concerned with the mental development of the child. The authors make it clear that there are many unsolved problems in the field of mental testing and that much work still remains to be done.

THE HUMAN FOOT

The Foot. By Norman C. Lake, M.D., M.S., D.Sc., F.R.C.S. Third edition. (Pp. 432; illustrated. 15s.) London: Baillière, Tindall and Cox. 1943.

Mr. Lake's book has not undergone any drastic changes during his preparation of a third edition. The most significant changes are the closer attention given to those foot conditions which are seen more frequently in wartime, such as those associated with cold, damp, or excessive strains.

True frost-bite is stated to be a much rarer condition than trench foot, but with the present tendency to rapid movement in military tactics, compared with the more static methods in the last war, the incidence of trench foot is likely to fall. On the other hand, true frost-bite has become more common as the number of high-flying airmen increase. The reference to foot strain in industry and military service is important. Long hours of standing on a concrete floor bring many industrial workers to their doctors or to hospital, where this general excessive strain on a static foot is found to be a frequent cause of disability, though aggravated in many cases by more obvious deformities such as flat-foot. The soldier who complains of pain in the foot with no obvious cause may well respond to a more gradual introduction to "foot-slogging" than is customary.

Without doubt this is an exceedingly useful book for the doctor in general practice and the orthopaedic house-surgeon, and it should help the regimental medical officer to keep the Army soundly on its feet.

TECHNIQUE OF PASTEURIZATION

Pasteurization. By Harry Hill, F.R.San.I. (Pp. 152. 10s.) London: H. K. Lewis. 1943.

Much has already been written about the public health aspects of pasteurization, and convincing evidence has been brought to show the need for heat treatment of milk in this country if a great deal of unnecessary disease is to be prevented. What is required now is a small book on the technique of pasteurization for the use of plant supervisors and operators, for medical officers of health and sanitary inspectors, and for laboratory workers in the public health field The small volume by Mr. Harry Hill entitled *Pasteurization* goes some way towards meeting this need. The author sets out to convince the reader of the necessity for pasteurization, and then goes on to consider the various processes by which pasteurization of milk may be effected. Neither of these aims is achieved with complete satisfaction. The discussion on the public health aspect is too condensed to be convincing, and the description of pasteurization technique is too general to be of much help to those who already have some knowledge of processing but would like a great deal more. It is curious that the author should have attempted this part of his task without including a single diagram, chart, or photograph in the book, and only three tables. Everything is left to the reader's imagination—the appearance, size, and lay-out of pasteurizing equipment, the internal structure of rotary pumps and of plate and tubular heaters, the design of the different types of bottle-washing machines, the relation between temperature and creaming capacity, and numerous other practical points for which simple textual description is inadequate. However, for those who want merely a general idea of the methods by which milk is pasteurized the book will be found of value.

Notes on Books

Mr. H. C. RUTHERFORD DARLING'S handbook *Surgical Nursing and After-treatment*, first published 26 years ago, has now reached an eighth edition (J. and A. Churchill; 12s. 6d.). In his preface the author acknowledges the co-operation of his colleagues at the Prince Henry Hospital, Sydney, for many hints and suggestions in revising his text and bringing it up to date. Admittedly surgical nursing is best learnt by actual experience in a well-equipped modern general hospital; but practical knowledge needs reinforcement, and the purpose of this little book is to elucidate the details and to reduce the duties of a nurse to a state of orderly sequence, so that practice and theory may be integrated. Much of the information and guidance in its 660 pages will prove helpful to surgical dressers and practitioners, as well as to the nurses for whom it was written.

Two sanitary inspectors, Mr. H. HILL and Mr. E. DODSWORTH, have collaborated in preparing a handbook for public health students entitled *Food Inspection Notes* (H. K. Lewis and Co.; 6s.). It is designed as a brief guide to present-day knowledge of the subject, stripped of all superfluous matter, and within its limitations should prove useful for revision before the candidate takes his examination. The legal aspects of the matter are not touched on.

Preparations and Appliances

PLASTICS IN SPLINT-MAKING

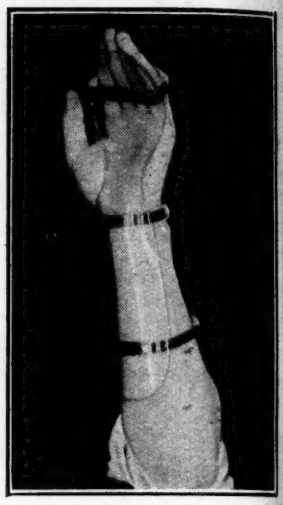
Mr. JAMES P. CAMPBELL, M.B., F.R.C.S.Ed., deputy surgeon-in-charge, Harlow Wood Orthopaedic Hospital, near Mansfield, Notts, writes:

Splint-making is a craft which is as old as the medical profession itself and is one which has a wide appeal, as it affects the work of the general practitioner and specialist alike; none more so than the orthopaedic surgeon, for whom it is an integral part of his daily work. The use of any new material in splint-making is therefore bound to arouse interest even though it is still in the experimental stage. A final statement as to its durability cannot be made until it has been put to the test of time. Plastics in one or other of their many forms may prove a valuable addition to those materials already in use in splint-making.

Any material which is light in weight, is easily moulded, and which retains its shape when fashioned is worthy of a trial in the manufacture of splints. "Perspex" (methyl methacrylate)—one of the group of plastics—is such a substance which has the added advantages of being non-absorbent, practically non-inflammable, and (so far as we know) non-irritant to the skin. It is as transparent as glass and casts no shadow, nor is there any distortion to *x* rays. In addition, it is tasteless and odourless and resists both acids and alkalis.

Perspex is supplied in sheets of varying size, and the samples under trial were 3/16 in. in thickness, but other thicknesses can be obtained. It can be cut and drilled when cold, but it is important to remember that bending and moulding can only be performed when it is in the plastic state, and this requires a temperature approximating that of boiling water. If attempts are made to bend it at too low a temperature it will crack, and cracks cannot be repaired, as one might imagine, by heating to the plastic state.

General Principles.—Perspex can be cut in simple shapes with a hack-saw, but curves are more easily cut with a fine-bladed fretsaw, and a little water will be found helpful when the saw is being used. Holes can be drilled with an ordinary drill such as that used in wood-work and metal-work. The edges can be rounded off with a file and if necessary smoothed with glass-paper or emery cloth. Moulding requires preliminary heating either in boiling water for two minutes or in front of a fire until the material becomes malleable. It can then be readily moulded to the desired shape, which must be retained until it is cool, and this may be hastened by immersion in cold water. Further adjustments in shape can be made by re-heating. Simple curves can be moulded by hand, but the more complex forms require a cast similar to those used in making block-leather splints. Two pieces of perspex may be cemented together with diakon cement, which is perspex in liquid form. The edges must be clean and fit closely; the cement is brushed on one half and the other quickly applied; light pressure is maintained for 12 hours in a warm temperature to allow the cement to set. The splint may be polished with special polishes—No. 1 and No. 2—to remove minor scratches and to give a finish to it.



Method of Splint Construction.—In making a splint from perspex the pattern in paper is first drawn on the paper-covered perspex and then cut out with a fretsaw. The edges are rounded off with a file and glass-paper. The perspex is then heated evenly throughout its length over a stove until it is soft and plastic. It is then bandaged to a cast and immersed in cold water for 1/2 to 1 minute. This process is repeated until the desired shape is obtained. Small drill-holes are then made at the appropriate levels for the straps to retain the splint in position. Before finally fixing the buckles and straps the splint is polished.

Splints made from perspex have certain advantages over metal and leather splints. They are lighter and less conspicuous; they are more hygienic in that they can be removed and washed with soap and water; they require less labour in manufacture as the finishing is more simple and padding or lining is unnecessary. The material can be used over and over again by simply heating and remoulding.

Perhaps its most useful field will be in cases of anterior poliomyelitis, as the lightness of the splint is of great importance in this condition, particularly in childhood, and perspex fulfils this need. This also applies to some types of nerve injuries such as wrist-drop of radial nerve paralysis. A further group of cases in which perspex splints may have definite advantages is where soiling is liable to occur. This applies to corrective splints for the lower limbs in infants and young children in the treatment of the deformities of rickets and where splints are required in incontinent patients.

The cost of splints made from perspex, so far as this can be estimated, may well be less than that for similar splints in metal, as the material costs almost the same and time in finishing a perspex splint is much less than that required to line and finish a metal splint.

B. Whitehall (*Johns Hopk. Hosp. Bull.*, 1943, **73**, 265) records a case of severe cerebral malaria due to *Plasmodium falciparum* in a soldier aged 25. Recovery followed the use of quinine intravenously. The urine and blood-pressure findings agreed with those of acute glomerular nephritis.

BRITISH MEDICAL JOURNAL

LONDON

SATURDAY JANUARY 29 1944

PULMONARY EMPHYSEMA

Pulmonary emphysema and its ally chronic bronchitis are two of the most distressing conditions with which a physician can be faced. The inexorable progress of the disease induces a despair in both patient and doctor, which the weary repetition of "mist. tuss. expect." does little to relieve. Many a physician thinks himself fortunate if he can divert such a patient to his houseman or to the casualty officer. So when a physician-physiologist turns his attention to such a chronic disease as emphysema, as Prof. R. V. Christie does in his Goulstonian Lectures published in this and last week's *Journal*, we welcome the brave attempt to throw new light on an old problem.

Laennec[1] gave the first detailed description of pulmonary emphysema. He considered the primary condition to be a bronchial catarrh which caused partial obstruction of the smaller bronchioles: as inspiration was a more powerful act than expiration, air tended to be trapped in the alveoli, which filled out and burst. Gairdner[2] and others, however, contended that the primary condition was some disease causing collapse or fibrosis of parts of the lung, which was followed in turn by distension of the remaining alveoli. Freund[3] and Loeschke[4] believed that spinal deformity and fixation of the chest in a position of inflation caused emphysema, and operations to correct this deformity were devised. Degenerative changes in the lungs, both vascular and nutritional, have also been called in to explain emphysema. A common assumption is that emphysema is associated with over-distension of the lungs, and many a student has treasured in his mind a picture of the purple-faced trombone player rending his alveoli in a mighty effort. But Christie states that the emphysematous lung only appears distended when seen at necropsy, because, unlike a normal lung, it does not collapse. He shows that the residual air in emphysematous subjects is increased only slightly, and that the lungs are often no more distended than normal lungs in moderate inspiration. As Christie has pointed out previously,[5] no man need hesitate to join a brass brand for fear of emphysema.

Sir John Floyer in 1698 noticed that the emphysematous lung lacks elasticity. Writing of broken-winded horses in his *Treatise of the Asthma*, he described the "rupture and dilatation of the bladders of the lungs" so that "the air blown into any lobe will not be expelled thence by the natural tone of the muscles of the bladders." As Thomas Watson[6] commented nearly 200 years later: "Really this is a capital piece of pathology for the seventeenth century." Loss of the normal pulmonary elasticity forms the basis of Christie's theory of the mechanism of emphysema; he considers it the most important abnormality in the emphysematous lung. He found the intrapleural pressure in several emphysematous subjects to be raised and often atmospheric, even at the end of inspiration. The elastic tension of the lung produces the normal negative intrapleural pressure, the abolition of which Christie takes as an indication of a loss of pulmonary elasticity. The weakened inelastic alveoli tend to dilate. The resulting enlargement is most noticeable close to the surface of the lung, where the pull of the chest wall and diaphragm is greatest: emphysematous bullae are commonly situated in this area. Expiration, as is well known, is largely a passive movement produced by the elastic recoil of the lung, and if this is abolished the chest would expand, owing to the overaction of the muscles of inspiration, thus producing the emphysematous deformity. Christie has shown that such expansion actually occurs when the pulmonary tension is abolished by an artificial pneumothorax.

The oxygen saturation of the arterial blood in emphysema is diminished in proportion to the severity of the disease and can be increased by giving oxygen (Meakins and Davies[7]). The ventilation in the lungs is therefore clearly deficient. The blood CO_2, on the other hand, is much increased and so is the alveolar CO_2; this suggests that a free gaseous interchange can take place through the alveolar walls. What is the explanation of this diminution of the oxygen and increase of the carbon dioxide in the blood and alveolar air in emphysema? Defective respiratory mechanism does not cause it, for the volume of the tidal air is not decreased. Insufficiency of functioning lung causes decreased CO_2 elimination as well as decreased O_2 absorption: so this cannot be blamed. Nor can one blame poor ventilation of the alveoli from distortion of the terminal bronchioles: gaseous exchange is so quick that such a defect could not impair the exchange of gases. Christie's explanation is that the inspired air tends to be drawn into the dilated and avascular alveoli and bullae at the expense of the more normal alveoli, which come to depend almost entirely on diffusion for their ventilation. Such an unequal ventilation in emphysema would account for both the insufficient oxygenation of the blood and the failure to eliminate CO_2 from the lungs. The dyspnoea is clearly due to the high blood CO_2, which even a much-increased bicarbonate reserve fails to prevent.

As to the actual cause of emphysema, Christie contends that the repeated strains on the alveolar walls during coughing or the obstructed breathing of asthma account for the loss of pulmonary elasticity. He draws attention to the almost invariable association of chronic bronchitis or asthma with emphysema, and to the fact that several workers have recorded increased alveolar pressures in these conditions. This theory, he admits, does not explain the occasional instances of emphysema in which there is no evidence of bronchial disease, but it helps to clarify much that was hitherto obscure. Nevertheless, the older theory of chronic obstruction will probably continue to have its supporters. As long ago as

1 *Traité de l'Auscultation Médiate*, 1819.
2 *Mthly. J. med. Sci.*, 1849, **9**, 242.
3 *Dtsch. med. Wschr.*, 1913, **39**, 603.
4 *Handb. spez. path. Anat. Histol.*, Berlin, 1928.
5 *Edinb. med. J.*, 1939, **46**, 463.
6 *Principles and Practice of Physic*, 4th ed., London, 1857.
7 *Respiratory Function in Disease*, Edinburgh, 1925.

1885 Brown-Séquard[8] noticed that when stimulation of the mid-brain produced bronchial spasm in a dog the alveoli dilated. This observation has since been confirmed, and it has been suggested that bronchial spasm may be present in all cases of emphysema, even when it is not clinically demonstrable. Kountz and Alexander[9] have shown that administration of adrenaline improves the vital capacity of patients with uncomplicated emphysema, as well as of those with bronchitis and asthma, thus supporting this suggestion. Chronic obstruction would also explain the intrapleural pressure changes. Christie, as Cabot,[10] Davidson,[11] and others before him, has found emphysema at the necropsy of patients who showed none of the classical signs of the disease during life. The barrel chest—the commonest diagnostic criterion—may also often be a postural deformity unassociated with emphysema. Of more value is the history of increasing dyspnoea on exertion in a chronic bronchitic or asthmatic.

A quick glance at these investigations might lead the practising doctor to think that they are largely of academic importance. But they point to three valuable methods of treatment which, though not new, are often neglected. Christie emphasizes once more the value of ephedrine—often very effective in relieving the dyspnoea of emphysema even when bronchial spasm is not evident. Respiratory exercises designed to improve diaphragmatic breathing and mobilize the lower chest are also most useful, especially in cases of marked thoracic deformity. Finally, Christie agrees with Kountz and Alexander in recommending the use of an abdominal belt which helps to raise the diaphragm. Application of these practical measures should ensure at least that the emphysematous patient leaves hospital with less dyspnoea, a more mobile chest, and a better-corseted abdomen.

A NEW BRITISH JOURNAL

This week sees the publication of the first number of the *British Journal of Industrial Medicine*, whose advent was heralded in these columns in an annotation a short while ago.[12] It owes its origin in the first place to that active group comprising the Association of Industrial Medical Officers, which was formed in 1935 to bring together medical men employed whole-time in various branches of industry so that they could pool their experiences and forward their common aims. As the editorial pen records, the new association got much valuable guidance from Sir Wilson Jameson, who was then dean of the London School of Hygiene and Tropical Medicine. The association grew rapidly and now has more than 300 members, to whom the new journal will mostly look for its inspiration and original work. The *B.J.I.M.*, which is being published from Tavistock Square by the British Medical Association, will, it is hoped, play more than a passive part in furthering knowledge in the field of industrial medicine. Its very existence should stimulate those whose professional work brings them into contact with industry to look at old problems with new eyes, to find fuller answers to old questions, and to start asking new questions, the answers to which will come out in work that can with truth be described as original.

Although, like the other three special quarterly journals published by the B.M.A., the new quarterly is first and foremost scientific in nature, the list of contents of No. 1 shows that the Editors are not afraid to provide that good mixed diet held to be the height of nutritional excellence. This pleasant foretaste of editorial policy convinces us that the *B.J.I.M.* will appeal to a much wider audience than the relatively small number of men now engaged whole-time in industrial medical practice and research. As Lord Moran says in his most apt foreword, written in a style which has his hall-mark: "The firms that in bulk bear the main burden of the day are generally too small to have a whole-time works doctor. They will look for help to the general practitioner, and there is nothing in the nature of industrial medicine which is beyond the competence of a sound practitioner." Lord Moran agrees, of course, that special training is required for factory work and that an industrial doctor must be familiar with working conditions and industrial hazards: "But first and last he must be a good doctor. If he is, all the rest will be given unto him."

No. 1 of the *B.J.I.M.* can certainly give him a good deal. It starts off with an article by Prof. Major Greenwood entitled "The Evolution of an Industrial Society." The author in one sentence shows how much of what we may call "industrial medicine" is simply part and parcel of medicine as a whole: "Had our ancestors wholely neglected the evils of life *in* factories, left young persons without the protection of laws and inspectors, disregarded extra-domestic evils, *but rehoused the people* [our italics], I think the rate of mortality would have begun to fall a century sooner." In one sense, then, industrial medicine is domestic medicine writ large, as we are reminded by the inclusion in this issue of the *B.J.I.M.* of articles on heating and ventilation by Dr. T. Bedford, on nutrition and the industrial worker by E. H. Capel, on some foot faults related to form and function by Mr. W. Sayle Creer. Heating and ventilation and nutrition—housing and food—are after all the first problems of domestic medicine and of social medicine. Some aspects of these problems may well first be studied in the factory and the workshop, and the lessons learnt be applied afterwards to the home—the medical province, as we like to believe it, of the family doctor.

There are of course the special problems of industry, more especially those of toxicology, as the article by Alice Stewart and L. J. Witts on chronic carbon tetrachloride intoxication reminds us. But perhaps the largest scope for inquiry by the medical man who has a whole-time or part-time special interest in industrial medicine is provided by the study of the individual undergoing the various stresses and strains of any given environment. How, for example, will the woman whose overactive thyroid has been removed stand up to her job in the textile factory? The factory and the workshop also give the doctor and the welfare worker an opportunity to find out how people live and to give them that instruction in personal and communal hygiene which this war has so clearly shown to

[8] *C. r. Soc. Biol.*, Paris, 1885, **37**, 354.
[9] *Medicine*, 1934, **13**, 251.
[10] *Physical Diagnosis*, Baltimore, 1927.
[11] *Proc. roy. Soc. Med.*, 1936, **29**, 1315.
[12] 1943, **2**, 584.

be necessary. What can be done in the way of investigating health and disease in a large group of employed people is a lesson that we in this country have learnt from the Post Office Medical Branch, under its recent chief medical officer, Sir Henry Bashford, who saw this first number of the *B.J.I.M.* to press and who contributes an article to it on some aspects of sick absence in industry. His appointment as Medical Adviser to the Treasury made it impossible for him to continue in his post as Chief Editor, in which he was succeeded by Dr. Donald Hunter before the first number came to light. We would, however, like here to record that he has been the moving spirit behind this new venture. The last article in No. 1 is contributed by a distinguished representative of industrial medicine in the U.S.A., Dr. Orlen J. Johnson, who writes on medical service in industry in the United States. This shows that the new journal, though British, is not going to close its columns to articles from men working in other countries on the common problems of industrial medicine.

AN INCREASE IN DIARRHOEAL DISEASES

Those who study the epidemiological tables of the *Journal* find almost every week something to stimulate their interest. Thus in the issue of Jan. 15 they noted with some excitement that notifications of diphtheria for the English great towns had dropped below 500 in the last week of 1943, which suggests that prophylactic immunization is already having an appreciable effect. On the other side of the picture, as the analysis on page 125 last week showed, is a recent increased incidence of scarlet fever, and—much more serious—a sharp rise in the war years of notifications of dysentery and of deaths from enteritis in children under 2 years of age. Indeed in 1943 more than three times as many children died of enteritis than of diphtheria in the large towns of England and Wales. It may be that a proportion of cases certified as dying of enteritis should be attributed to other causes, but this error is not likely to vary much from year to year, and does not explain the steady trend upwards of deaths from 1,812 in 1940 to 2,569 last year. An even more striking increase in the notifications of dysentery (from 2,843 in 1940 to 7,772 in 1943) can only partially be explained by a greater awareness and better diagnosis of this, now endemic, infection. These increases in diarrhoeal diseases at a time when the infant mortality rate is improving must give us pause.

Dysentery in this country is a mild disease of high infectivity, and if, as is often stated, its incidence reflects the standard of personal and public hygiene in the community we must suppose that there has been some falling-off in these standards since the war began. A variety of reasons suggest themselves: a great increase in communal feeding, the multiplication of day and residential nurseries, labour shortage interfering with the cleanly handling of food and milk (a number of milk-borne outbreaks of dysentery have lately been reported), and probably mass movements of troops and civilians. But we need also to consider if there has been some lowering in the host-resistance due to improper feeding or physical or mental fatigue. Dysentery is mainly spread by the contamination of food with soiled fingers, but too often the mildness of the disease means that the culprit cannot easily be detected. Add to these missed cases the contact and convalescent carriers, now shown by selective culture media to be much more common than was formerly believed, and it is understandable how difficult a disease dysentery is to eradicate. Only a high standard of personal hygiene in the community, and particularly among food-handlers, will control its spread, and propaganda to this end is urgently needed. The Food and Drugs Act (1938) gives the M.O.H. new and wide powers in dealing with premises and personnel where food is sold.

Infantile diarrhoea, if less prevalent than dysentery, is a much more fatal infection, and ranks next to neonatal deaths and pneumonia as a cause of infant mortality. Its aetiology is complex, but its ravages could be largely prevented if a majority of mothers could or would breast-feed their babies for 6 to 9 months. Here again enlightened propaganda[1] and much concerted effort by family doctor, ante-natal and child welfare clinic, maternity department, and the mothers themselves would encompass a great saving of lives. While some 80% of mothers delivered in institutions and 95% of those confined at home breast-feed their babies for the first few weeks of life, the proportion has dropped to 50% or less at 3 months and 30 to 40% at 6 months. With a great increase in institutional midwifery much depends on the nursing staff of the maternity unit in establishing satisfactory breast-feeding, and no unit is doing its job properly that cannot discharge about 90% of wholly breast-fed babies. Too often, however, much painstaking effort in the maternity home is undone when the mother, returning after 10 days to the worries of a household, either cannot or will not continue with breast-feeding. In this she may be aided and abetted by her family doctor, who is sometimes too ready to recommend artificial feeding when a little encouragement and advice would ensure continuance of Nature's way. Nor are welfare clinics entirely blameless, especially when their posters proclaim the virtues of this and that brand of dried milk. It must not be forgotten, however, that infantile diarrhoea affects principally poor-class urban children, so that factors other than artificial feeding operate. For example, are the salvage campaign and the collection of pig-food, which helps to breed flies, a contributory cause of the greater prevalence in the war years? Is there a falling-off in the cleanliness of milk due to less careful handling and poorer distribution? Are children with minor digestive upsets too readily certified and too easily admitted to hospital, and is hospital cross-infection more prevalent because of shortage of nurses? Again, outbreaks of neonatal diarrhoea, with its appalling mortality, have been more common in recent years, and this infection, too, occurs mostly and in its severest form in bottle-fed babies. The attack on the whole problem of infantile enteritis seems to be along the lines of intelligent advice and propaganda for the mother about breast-feeding, more effort in maternity units to attain a 90% discharge of wholly breast-fed babies, and, most important, more home-help when the mother again takes up the onerous duties of housewife.

[1] See Rep. on Publ. Hlth. and Med. Subjects, No. 91: *The Breast-feeding of Infants*. 1943. (4d.)

INFANT MORTALITY IN BELFAST

The most notable feature of the mortality experience of the present century is the decrease in infant mortality, which has fallen by two-thirds in England and Wales. Striking as this reduction is, much lower rates have been attained by some countries. Infant mortality in the large towns of Great Britain has lagged behind the country as a whole. That conditions of life in big cities need not be associated with a severe mortality risk to infants has been demonstrated in America, where very low rates have been recorded—e.g., in 1942 the infant mortality of New York was only 29 per 1,000. It is obvious that there is ample room for improvement in the British rates, and it seems to be within the bounds of possibility that infant mortality in large cities can be reduced to at least 25 per 1,000. The city of Belfast is unfortunate in having one of the largest infant death rates in the United Kingdom. The deaths of infants in this city have fluctuated from 1 in 12 to as high as 1 in 8 in recent years, and judging by existing standards about three-quarters of these deaths were preventable. To put it another way: over 500 babies die yearly who apparently could be saved. An investigation into the various aspects of this mortality in Belfast has been made by Deeny and Murdock.[1] The data comprised the deaths between June, 1941, and June, 1942, and a random sample of one-fifth of the children born in the first six months of this period who survived the first year of life. Premature birth and injury at birth accounted for one-quarter of the deaths; another quarter was due to respiratory diseases, and one-fifth to diarrhoea and enteritis. The environmental indices—income and housing conditions—were related to infant mortality: the former had the larger association, but rather surprisingly no significant relation could be demonstrated between dirty homes and mortality. The deaths from diarrhoea and enteritis and respiratory infections formed a larger proportion of the total infant deaths in the lowest income group than in the highest. An attempt was made to determine the adequacy of the medical attention by examining the individual cases. Medical aid was sought as promptly in the illness that ended fatally as in non-fatal sickness. The authors found that parents were quick to call in the doctor, and the poor secured medical attention as early as those who were better off. Nutrition is of first importance in the rearing of healthy children, and the relation of feeding to infant mortality is discussed by the authors elsewhere in this *Journal*. The proportion of breast-fed babies was lower among the dead than among those who lived. The proportion breast-fed was practically constant in both groups of infants for the subgroups of income per head, number of persons per room, domestic hygiene, and for order of birth and cause of death. These factors had no significant influence in determining the proportion of breast-fed babies, and no relation was established between breast-feeding and mortality. The authors conclude that an increase in the proportion of breast-fed infants would not lower the infant mortality, and that the smaller proportion found among the dead was an effect rather than the cause of unsuccessful reproduction.

[1] *Ulster med. J.*, 1943, **12**, 117; and *Infant Mortality in the City of Belfast*, Statistical and Social Inquiry Society of Ireland.

SURGICAL SCIENCE MOVES FORWARD

The Scientific Report of the Royal College of Surgeons of England for the past academic year makes cheerful reading. There is a record of painstaking care of the surviving material from the Museum scattered widely over the Home Counties. Shortage of staff and difficulties of transport can be understood by most, but the attention and skill needed to conserve material from the depredations of mice, moulds, and men can only be appreciated fully by those responsible for the care of museums. It is encouraging to read of the plans made for collection of material in special fields by College representatives chosen for their interest in them. Donors have been numerous and their gifts many and valuable. The odontological collection of the Royal Society of Medicine has been given to the College as a contribution towards the future reconstruction of the Museum.

The College has decided on its future policy concerning the Museum. An opportunity is now presented to provide not only a unique collection of anatomical and pathological material but an educational instrument of untold value. Medical museums are essentially educational. The day has long passed when they were the repositories of the surgical triumphs of bygone surgeons and bleached specimens hallowed by a famous name. The historical value of the latter far outweighed their pathological interest. It is to be hoped that in the new Museum a place will be found for Hunter's specimens, a place where honour can be done to Paget and Lister and where specimens illustrating classical work will be on view. The greater part of the Pathological Collection it is hoped will grow into a national type-collection of pathology adequately documented with clinical histories and illuminated with histological detail—these for the specialist as his reference library. For the student who wishes to see pathological processes let us hope there will be a place. The anatomical series can never be reconstituted as Hunter formed it—a collection to illustrate the relation of form to function—but perhaps it will be possible to suggest somewhere the great idea which Hunter had and which was so fruitful both before and after Darwin. In this collection, too, the collections for the specialist will be necessary, and it is to be hoped that examples of superb prosectorial skill will be available for the student of human structure.

The Research Laboratories are obviously in battle dress. Experiments on the production of casein digests suitable for intravenous medication, the prevention of liver damage during antisyphilitic treatment, the treatment of infective hepatitis, and new surgical suture material are all of topical as well as of scientific interest. As reports on these various problems will soon be published they do not call for discussion here. More significant than the problems has been the way in which these laboratories and their staff have become responsible for a large-scale clinical experiment. It must be rare for a research laboratory to have available sixty hospital beds where new methods of treatment can be worked out (under proper supervision) and where there is continuous interchange of ideas between the hospital ward and the laboratory. A significant paragraph in the report states: "This year's work has made it abundantly clear that in future there should be available to those working in the laboratories easy access to a hospital ward which is dedicated solely to the study of disease. The constant stimulus which comes from team work is immeasurably facilitated by direct contact with and responsibility for the treatment of the sick." Perhaps in the near future some such research hospital might be established on or near the College site. With the existing Research Laboratories, and the imminent developments in the Department of Pathology close by, a research centre of unlimited usefulness would soon come to maturity.

We much regret to announce that Prof. W. W. C. Topley, M.D., F.R.S., late Director of the Division of Bacteriology and Immunology, London School of Hygiene and Tropical Medicine, died suddenly on Jan. 21, after attending a meeting of the Agricultural Research Council, of which he had been Secretary since 1941.

Reports of Societies

ORGANIZATION OF LUPUS TREATMENT

A joint meeting of the Sections of Dermatology and of Epidemiology and State Medicine of the Royal Society of Medicine was held on Dec. 16 to discuss the organization of the treatment of lupus vulgaris. Dr. A. C. ROXBURGH presided. The subject was introduced by Dr. F. S. AIREY, who said that there was no true conception of the incidence of lupus in this country, its distribution, the facilities available for its treatment, their adequacy, the extent to which those afflicted availed themselves of the opportunities for treatment, and the success of the measures adopted. He suggested an incidence of one case per 1,000 of population, which would mean 45,000 lupus sufferers, and would justify the establishment of local treatment centres. Just as public health authorities had tackled diphtheria, so propaganda should be used to persuade lupus patients to seek treatment at an early stage. Lupus was a sociological as well as a medical problem.

Sir HENRY GAUVAIN (in a paper read in his absence) said that there should be recognized accessible centres where cases of suspected lupus could be sent for diagnosis. Severe cases of lupus in children should be treated at a resident hospital school, and adults seriously affected, in colonies. Correspondence in the *British Medical Journal* had evoked enthusiastic testimony to the Finsen-Lomholt lamp. Its drawback was its expensiveness. He believed that, Denmark being now in enemy hands, it would be legal to undertake the manufacture of the lamp in this country. But it was deplorable that Finsen treatment should have become a fetish.

Dr. A. BURROWS praised light treatment for its efficacy. Localized patches of lupus were ideally treated at Finsen light centres; more extensive cases could be successfully treated by light, but the applications might have to be protracted over a long period. It used to be said that lupus patients did not suffer from other forms of tuberculosis, but that idea had been exploded. In many cases the patients could carry on their ordinary lives while under treatment, but segregation was sometimes desirable. Segregation had the advantage of ensuring hygienic surroundings and aseptic dressings; its drawback was the psychological effect. The proper method of organization would be *ad hoc* centres with some residential accommodation for special cases over short periods. He thought that about ten centres in England would be sufficient.

Incidence of Lupus Vulgaris

Dr. F. R. G. HEAF said that lupus vulgaris was a comparatively rare condition. In the five years 1931–5, out of a total of 3,624 non-pulmonary tuberculosis cases treated at L.C.C. centres, 59 were of lupus of the skin. In 1938 in Lancashire 2,032 cases of all forms of tuberculosis were notified, of which 805 were non-pulmonary, and the skin cases of lupus numbered 26. On the Lancashire registers in 1942 the number of active cases was 191, and of quiescent cases 85. In Manchester in 1940 there were 15 new notifications and in 1941 17. As treatment was costly and called for a specially trained staff, it should be located in centres which served at least a population of 5,000,000. The establishment of such centres called for regionalization and for co-operation between local authorities. He estimated that there were annually about 10 new cases of lupus per 1,000,000 population, and that five beds per 1,000,000 were required, with out-patient facilities to treat approximately 50 cases per 1,000,000. If there was much disfigurement, permanent residence at an industrial settlement like Papworth would have to be considered when the activity of the lesion had been overcome.

Dr. HENRY MACCORMAC said that he was under the impression that tuberculosis of the skin was a notifiable disease, as were other forms of tuberculosis, and therefore there should be official records of the number of cases. He believed it was an extremely rare disease, though that was no reason for neglecting it. Any attempt to regiment people into institutions would create great difficulty, but it might be possible to establish a system of occupational centres like that in Denmark. Lupus had a geographical distribution. It was common in Scandinavian countries and, he believed, unknown in Australia.

Dr. W. J. O'DONOVAN said that if lupus vulgaris was a problem of any size and had been neglected it would declare itself in the reports of the medical boards. In the whole of one Command, covering a quarter of the country, in which all difficult contagious conditions were referred to him, he had seen since the beginning of the war only 4 cases of lupus vulgaris, and those were slight. Up to the outbreak of war this was a diminishing problem, thanks to the greater care of public health, food inspection, and smoke abatement. When he began to assist Dr. Sequeira at the London Hospital there was a multitude of horrific cases, but these diminished as the years went on, while the number of early cases sent by school medical officers increased. In a paper in 1925 Sequeira said that the Minister of Health had conferred upon local authorities ample powers to set up clinics, and he believed these were sufficiently used in the areas in and around London. The key to the lupus problem was accurate initial diagnosis. It took him five years, during which he saw lupus daily in numbers of cases, before he was positive as to the state of the skin of the patients who came under treatment. Lupus was a manageable problem. He considered that the treatment at the centres could be applied, under a skilled and experienced sister, by nurses of the category familiar in the Army as V.A.D.s. At St. Louis he noticed that many of the nurses and hospital attendants had themselves suffered from lupus, and such people, if educated and courteous, should be the very best to persuade other patients to submit to the necessary treatment.

No Large Public Health Problem

Dr. A. M. H. GRAY said that lupus was not a large public health problem, and therefore had not been dealt with to any considerable extent from that point of view. At the same time, dermatologists would feel that it was only right for some active steps to be taken. The problem with adults was more difficult than with children, for some of them were unable to obtain work. Some arrangements for occupational therapy were desirable. Dr. Heaf had suggested that the treatment should be centred on general hospitals with teaching schools, but in London this would mean that the number of cases at each hospital would hardly be adequate from the teaching point of view. He would prefer to see cases of lupus transferred to one or two centres where treatment could be organized on a fairly large scale. Dr. J. A. H. BRINCKER pointed out that according to Lomholt the incidence of lupus in Denmark was about 1 in 1,000 of population. An investigation in London following upon Lomholt's visit ten years ago revealed very few cases. Dr. J. T. INGRAM said that in Leeds during the last fifteen years, from an area having a population of about 2,000,000, there had been 559 cases of lupus attending the out-patient department. Of this number 138 for one reason or other had had no treatment. Of the 421 cases treated 324 were apparently cured, and in 175 of these the cure had persisted for from five to ten years. About 27 cases out of the total had been marked down as incurable, but any case of lupus returning to adverse psychological, nutritional, or environmental conditions was liable to relapse. Colonization was necessary for only a very small proportion of cases.

Dr. R. T. BRAIN said that many physiotherapists appeared to be frightened of ultra-violet light. He had often referred a patient with lupus to a local light centre, and had been told that he could have two or three treatments for a week and then must wait for two or three months. He could not understand that attitude. It was important, of course, that the patient should not have latent phthisis, and it was the custom to refer patients to a clinician for examination of the chest. The kidneys also should not be forgotten, because a damaged kidney would be disturbed by the intensity of the radiation. But these conditions excluded, a patient could be given a very high concentration over a long period. Sir WELDON DALRYMPLE-CHAMPNEYS said that he would have liked to hear a little more about the competence of the general practitioner to make a provisional diagnosis of the disease. A certain amount of extra instruction in this disease should be given to medical students—enough at all events to enable them to suspect a case of early lupus and refer it to an expert for diagnosis.

Correspondence

Research in Ophthalmology

SIR,—I have read with interest the editorial entitled "Research in Ophthalmology" in your issue of Oct. 2 (p. 426), and while this undoubtedly merited a critical reply from some senior member of the profession, so far none has been forthcoming. One must assume that the reasons advanced for the undoubted paucity of basic ophthalmic research during the past decade in this country, which you so rightly deprecate, have the tacit approval of ophthalmologists. None has seen fit to join issue with you when you attribute a large measure of the fault to those junior ophthalomologists who "slip with such ease into the routine work of well-paid refraction clinics and thus side-step junior hospital appointments with their facilities for research."

This is, however, a most biased picture of the actual situation. In the first place it must be insisted that the causes which have operated to bring about the present unsatisfactory position reside at a much higher level in the ophthalmological heirarchy than the humble, overworked, and certainly underpaid ophthalmic clinical assistant and refractionist. The eye-hospital junior surgical appointments, with or without the facilities for research which you mention, are far too few to go round, so that a large number of practitioners genuinely interested in the academic side of the specialty are grounded at their particular level with little hope of advancement and with hardly any encouragement from above. For the most part they are excluded from the general work of the hospital and confined almost rigidly to the routine of the overcrowded out-patient departments. Their lot is one of discouragement and stultification, and until this pernicious system in eye hospitals receives its final quietus (and no movement to make an end of it is yet discernible from those responsible) it seems a pity to saddle this almost inarticulate stratum of the ophthalmic world with any major share of liability for the poor output of ophthalmic research.

However, the essential reason is the one you mention—"that the time is past when advances in knowledge can be attained by relatively simple methods by a single investigator," and particularly is this true in eye work. Surgical ophthalmology has already reached a very high stage of development, and medical ophthalmology can only advance in step with advances in general medicine as they occur. In fact, therefore, it is to medical ophthalmology that we must in the future devote the major part of our effort in ophthalmic research; but so long as ophthalmology is regarded as primarily a surgical specialty, and its academic associations continue to be inspired by this conception, the dearth of research is likely to continue.

For the past few years a section of the ophthalmic profession with these matters in mind have been endeavouring to establish a Faculty or College of Ophthalmology, and it is likely that in the near future their efforts will meet with success. Such a development is likely to result in a greater general level of interest and co-operation in ophthalmic research throughout the country than the isolated local establishment of professorial chairs attached to various universities and ophthalmic institutions, which, though laudable developments in themselves, are too restricted in nature to exert any widespread or rapid influence on ophthalmic education and research generally.

It is currently accepted throughout the ophthalmic profession that a regulated portal of entry into the profession which is nationally available and which is based upon high standards of both clinical apprenticeship and academic knowledge is long overdue. None of the present diplomas in ophthalmology satisfactorily equates the criteria mentioned, nor do the projected alterations in the F.R.C.S.Eng. (so far as one knows their details) seem likely to meet the requirements of the majority of ophthalmologists in practice.

It is the absence of such a central co-ordinating and stimulating institution in ophthalmology which is the most important single factor in the very marked meagreness of ophthalmic research in this country.—I am, etc.,

LIONEL M. GREEN,
President, Association of British Ophthalmologists.

London, W.1.

Consultant and Specialist Services

SIR,—The letter of the Presidents of the three Colleges fills me with some uneasiness at the methods employed. They refer to an existing committee of the Colleges, which consists, among others, of eleven representatives from the non-teaching hospitals. As a consultant and member of the staff of a non-teaching hospital I should be very interested to know how these representatives are nominated and elected; as a Fellow of the Royal College of Surgeons and a member of the Royal College of Obstetricians and Gynaecologists I cannot recollect ever having been given an opportunity to nominate or elect such representatives.

The next point on which I should like more information is the steps taken by the Colleges "to ascertain what consultants and specialists are available at the present time." What are the steps that are being taken?

But perhaps the most disquieting statement is that "the result of this survey will not be published; it is for the personal guidance of the Minister." These appear to be unusual methods in a democratic country. How does a consultant know whether or not he has been put on this list or left out if it is all so secret? I sincerely hope that information may be published that will allay these misgivings.—I am, etc.,

H. J. MCCURRICH.

Hove.

Future of the Mental Health Services

SIR,—Last year, when it appeared a matter of urgency to present the Minister of Health with some considered ideas on this subject, the Royal Medico-Psychological Association, like certain other bodies, prepared its tentative recommendations. Now, with rather more breathing space, the association has set up a new planning committee, under the chairmanship of Brig. J. R. Rees, designed to represent every branch of the specialty. It is hoped that in each division of the association a local committee will meet to discuss and represent the views not only of the members of the R.M.P.A. but also of any, or all, psychiatrists in that area. The committee thus hopes to receive a number of considered plans, but, in addition, it will welcome all relevant suggestions put forward by any group or by individual physicians. The mental health of the nation is of paramount importance, and accordingly the future organization of the mental health services merits most careful thought.

All such communications can be sent to me as below, anonymity being preserved if desired.—I am, etc.,

W. GORDON MASEFIELD,
Hon. Secretary, Medical Planning Committee, Royal Medico-Psychological Association.

11, Chandos Street, London, W.1.

Returned Prisoners of War

SIR,—May a psychologist congratulate Major P. H. Newman (Jan. 1, p. 8) on his statement of the difficulties of the returned prisoner of war. It might be criticized as only naked-eye pathology, but no claim is made to anything deeper; psychiatrists welcome his first-hand experience and observations, and what might be a defect becomes an advantage when the object is to appeal to the ordinary doctor to recognize and understand the condition of these men. Yet my experience in the treatment of ex-Service men after the last war leads me to amplify or qualify some of his statements. Thus the choice of escaping and sabotage as one way of leading a constructive life is too simple a view, even when qualified by the remark that the choice depends upon temperament. I believe it is technically the duty of every prisoner of war to try to escape, but when I listened to a radio account of a man who succeeded after four attempts, though the victim of savage punishment and the witness of the shooting of a fellow venturer, I recognize his state as probably identical with that of other men whose repeated efforts to escape depended upon a pathological compulsion that persisted in other form—a tendency to fugues, for example—after return to civil life.

A more useful broadcast would be extracts from Major Newman's account of the effects of the deprivations and frustrations of the men behind the barbed wire.

Major Newman, perhaps because of the surgical outlook that persistently regards nerves as corporeal structures, avoids that thought-stifling witch word *neurosis*, though he falls down once on *neurotic*. This makes more surprising his view that individual psychological treatment for any returned prisoner of war is "debatable" because "it may carry with it a public acknowledgment of mental abnormality, which must at all costs be avoided." This reminds us of, "He's not mental, doctor; it's his nerves," and is a poor reason for depriving the men of treatment that is now available for civilians in psychiatric clinics all over the country. I can assure Major Newman that the difficulty he pictures is quite familiar, and the solution is to remove the stigma from "mental." To-day I saw a letter from an ex-N.C.O. with 3½ years' service in the R.A.F., invalided out for what he says was "anxiety psychoneurosis," who asks for treatment, and writes, "I find life very hard at the moment as I am not doing any regular work," then describes how he worked for six months, but "became worse and came back home where I have been ever since, doing odd jobs of work for my parents." Would Major Newman deny this man individual treatment (supposing it is desirable) lest it might involve public acknowledgment of mental abnormality?

I hope he will carry on the good work and place himself on the side of the psychiatrists, some of whom were struggling on behalf of their soldier patients a quarter of a century ago—often in the face of somewhat sadistic moral judgments used as diagnostic criteria—for the same understanding and tolerance that he now seeks for the troubles of the men whose cause he champions.—I am, etc.,

London, W.1. MILLAIS CULPIN.

The Prisoner-of-War Mentality

SIR,—As a slight relief from the depression which Dr. Richardson Billings's letter (Jan. 15, p. 90) might have caused in the minds of relatives of prisoners of war in the Far East, I should like to mention that in the beginning of this month I received from my elder son, Major G. F. Harrison, R.A.M.C., who is a prisoner of war in Camp A, Hong Kong, three postcards, dated respectively March 14, July 11, and Sept. 12, in one of which he said: "We have a very good library, and, when there is time, I am sampling the contents with pleasure." In another, after mentioning that he had read the whole of Shakespeare's works, he said he was glad to have the opportunity "to read stuff I never seemed to have time for before." Incidentally, in one card he said that he had seen in a local paper dated June 18, 1943, mention of an article of mine that had been published in the *Spectator* for the week previously. This quotation must have been derived from a German broadcast in which the article was mentioned during the week following June 11.—I am, etc.,

London, S.W.1. L. W. HARRISON.

Shock Therapy

SIR,—Dr. Winnicott's letter (Dec. 25, p. 829) should be taken not so much as a challenge to innovations in psychiatry but as a warning for us all to pause awhile and think seriously about the general direction in which medical progress is taking us to-day. We live in a world of "action and reaction." If a certain course of action is pursued long enough that is not favourable to or in keeping with the internal economy of the "organization" or "organism," some catastrophe will be precipitated. In broad principle this is an inescapable truth and is universally applicable: if we think in terms of nations the catastrophe will be revolution or war; if in terms of industry, a strike; if in terms of the physical body, physical illness; if in terms of the mind, neurosis or psychosis.

We know very little more about the actual acquisition of disease than our forefathers did, or the real reason why a person falls ill. True we know a great deal about bacteria, viruses, etc., and enormous advances have been made in controlling the growth of these organisms; but what has modern medicine given us to enable a man to say: "I am proof against disease" (unless of course he has been artificially immunized)? Is it really to the advantage of the human race that all our energies should be devoted to the "repair shop"? The more efficient we make the palliative side of medicine, the less urgent will be the need to look to ourselves, our habits of life, etc., as possible "courses of action" which are unfavourable to, or not in keeping with, the internal economy of the human organism.

Other contributors have ably pointed out that psycho-analysis does make an endeavour to understand why a person is ill as well as how. It is more than a mere therapy; it is a science based on the understanding of the interplay of conscious and unconscious forces. In judging the therapeutic properties of any "cure" we fall into error if we consider only the effect it has upon the symptoms of the illness and ignore the known pathology of the condition. In 1917 Freud outlined the psychopathology of depression (*Mourning and Melancholia*), and since then analysis has shown clearly that it can be regarded as a normal phase through which the child passes (though, of course, seldom observed as such). It would seem that the advocates of shock therapy will have to do one of two things: either think of the therapy in terms of what happens in the transference relationship with their patients by the introduction of fits, or else work out a new pathology in a physiological setting and demonstrate how the fit alters this structure.

It may not be without significance that nearly all the reports on shock therapy given by the administrators minimize the anxiety experienced by the patient and stress the harmlessness of the procedure. As a psychiatrist I have now had the opportunity of seeing quite a number of patients who have received this treatment at different centres. When telling me about the treatment they have nearly all stressed the terrifying nature of the procedure. This marked discrepancy of findings is, I think, most enlightening. It can be taken as a clue to the nature of the unconscious transference relationship initiated by the fit—one in which the aggressive component predominates, with its affect of hate and the reaction formation of terror; they are thus canalized and bound into this relationship and the patient is thereby freed. If this be true, then it would follow that the patient would repress the memory of his actual experiences and substitute some pleasing account for the benefit of the physician, and only later, to a sympathetic but otherwise disinterested person, can they tell the real facts. I would even go so far as to say that all reports given by these patients to any member of the staff of the hospital might be so biased as to be quite worthless, but would be very interesting as to what a patient can say and do under a certain set of circumstances which are not in keeping with his internal economy. This mechanism was clearly seen in a case of paranoia with delusions of persecution I was treating with insulin coma. He quite rapidly lost his delusions and substituted an ingratiating attitude. During the three months after conclusion of this treatment there was a gradual diminution of this "pleasing" attitude, and then a fairly sudden swing to the negative phase in which I was the predominating character in his delusional system of persecution.—I am, etc.,

Bishops Stortford. D. N. HARDCASTLE.

SIR,—With reference to Dr. Winnicott's letter in your issue of Dec. 25, which has just reached me, the views of one who has used electric shock therapy in private practice in addition to psychotherapy may be of interest. As yet I am in the dark as to *how* electric shock works, but I do know to some extent *what* it does. In general, it has every appearance of acting primarily as a powerful and prolonged general tonic, so marked that it will rejuvenate to the extent of removing the ravages of several years (plus anxiety signs) from a paranoiac without in such a case altering the ideas one whit. I consider that it does this through a stimulation of the pituitary gland, and an indirect further stimulation of the whole endocrine orchestra, easily seen in the frequent upset of the ovarian rhythm. Acting on this view I have recently used subconvulsive doses, giving two or three at a time, with a total result in suitable cases as good as if convulsive doses are given.

I consider that heavy dosage may produce further effects—possibly fracture of the physical associational fibres in the frontal area. In some cases this may be of value, when a

patient is unwilling or cannot afford to undergo a prolonged course of psychotherapy, and in which treatment the association tracts of unhappy childhood may be "leucotomized." After due warning to the patient that such treatment is a surgical operation, I have attempted this twice, once with apparent success.

In general, I try to reserve electric shock for the middle-aged depressive, whose state is of physical origin, and only give it on occasion in the course of a session of psychotherapy as a physical tonic, making it quite clear to the patients that it will make them "feel" better in body and give them more factual energy, but that it will not alter the "feelings" of their minds in the least, which must be worked through and made fully conscious in the ordinary way. I quite agree that giving it to a child smacks of barely unconscious sadism.—I am, etc.,

Dublin. L. F. DONNAN.

SIR,—As the result of analytical observations (using the method of analytical psychology) I am able to support Dr. Winnicott's statement; the process at work in shock therapy, from the patient's standpoint, is essentially akin to suicide, though, of course, actual suicide does not occur. The method of analytical psychology is different in many respects from that of psycho-analysis, so that it is a striking fact that two people using different methods arrive at almost identical conclusions.

I should like to point out that our view, derived as it is from the detailed study of individual cases, receives the strongest support from the reported efficiency of shock treatment for depressive cases, in which real suicidal impulses are common. If this conclusion be correct it would be extremely difficult for psychiatrists using shock treatment to see what is going on when the shock is applied, because they are participating in a method whose aim is to prevent the necessary information being revealed. Naturally these psychiatrists would deny the possibility of suicide coming into the picture at all. This has actually been demonstrated in your correspondence.

It is certainly bad psychiatry to use shock therapy as it is being used to-day, and the ethical question cannot be brushed aside in the cavalier fashion which some of your correspondents assume.—I am, etc.,

Nottingham. MICHAEL FORDHAM.

Significance of Dysphagia in the Psychopathic

SIR,—In the article describing a case of conversion hysteria by Dr. Ranyard West (Nov. 27, p. 676) there are several points in the case which I think are worth further explanation. Despite the extremely psychopathic history which the patient showed, may I ask if the diagnosis of myasthenia gravis was ever considered in this case? In the early part of the history it is stated that the patient could drink fluid in the morning, though not easily, but later in the day the fluid went the wrong way. This dysphagic symptom is extremely suggestive of myasthenia gravis, where muscular effort becomes increasingly difficult as the day progresses. We also know that in myasthenia gravis there are normal remissions of the disease which might account for the improvement of the patient with psychological treatment. The fact that the analysis took forty sessions seems to imply that the treatment took a long time to become effective.

Recently I have seen a case of a girl who was considered as a psychiatric problem for three years, whereas, in actual fact, she was a clear-cut case of myasthenia gravis. One psychiatrist had missed the myasthenic facies, and considered she showed the typical *belle indifférence* of the hysteric. The fatigue which she complained of was also put down as a neurotic symptom. It is true that in the early stages myasthenia gravis can be extremely difficult to diagnose, particularly differentiating it from a hysterical condition.

I do not say that Dr. West's case was one of myasthenia gravis, but certain points in the history are rather suggestive. I would also add that I consider dysphagia to be a very rare hysterical symptom.—I am, etc.,

M. CAMPBELL,
Squadron Leader, R.A.F.

The Mosley Incident

SIR,—Now that the conditioned release of Sir Oswald Mosley from detention under 18B has lost its sensational news value it may not be improper to consider those of its aspects which are of interest to the medical profession. They are two in number: one relates to herd psychology and the other to constitutional and ethical procedure.

Herd Psychology.—The "patient" in this case is a private citizen with a private citizen's rights, including that of entertaining and maintaining his own political faith. This country is at war with nations who contest that right and who do all in their power to suppress it, and it is largely to maintain what we believe to be an individual freedom of faith that we are fighting. But owing to the war it became necessary to prevent the patient from actively spreading his gospel without interfering with his freedom of faith and without any intention of inflicting punishment. A large proportion of the herd became confused in their reaction to the conditioned release of the patient, and showed by their behaviour and by their written and spoken word that his punishment by severe suffering or even by death was the object of their desire. And, as is not unusual, the herd instinct may be described in this instance as quite illogical and indefensible.

Constitutional and Ethical Procedure.—The patient, being ill, had a right to medical advice, and his medical advisers, strengthened by the opinion of eminent consultants, reported that his health and perhaps his life would be endangered by further detention. At this point a question arises which is one of constitutional and ethical importance. Has this patient also lost his right to professional secrecy in regard to the nature of his malady? And further, if he had not lost it, did he attempt to exercise it? The nature of the patient's malady and the treatment prescribed were publicly disclosed, whether with or without the doctors' consent we are unaware. At any rate the disclosure gave rise not only to uninformed and hysterical criticism in the Press but to ignorant and foolish questions in the House of Commons. In both instances a lamentable ignorance on the part of the public was brought to the surface. A doctor's responsibility is not only to treat his patient's local disease but to treat his patient as a whole.

We may hope that no further incident of this kind may happen, but we may justifiably ask for an authoritative statement in reply to the constitutional and ethical questions which I have raised. Without prejudging the issue I would suggest that the medical report given to the public would have satisfied all legitimate demands if it had contained only words to this effect: "In our opinion the health and perhaps the life of Sir Oswald Mosley will be endangered by his further detention."—I am, etc.,

Oxford. E. FARQUHAR BUZZARD.

Vulvovaginitis in Children

SIR,—In his article on the treatment of gonorrhoea in the female Brig. Osmond has necessarily given a restricted account of vulvovaginitis in children, but I regret that he has not stressed the importance of in-patient treatment under the best hygienic conditions. Many of these children come to us in a depressed state of general health, infested with vermin, and with impetigo and other skin diseases. A survey of admissions since 1939 shows that only 48% were found to be gonococcal before or after admission, so that the majority of vulvovaginitis infections under 10 years of age are due to other organisms—e.g., the staphylococcus, *B. coli*, streptococcus, etc.—and the primary lesion is usually found in a site other than the genital tract. Frequent bathing, good food, fresh air, and regular discipline do wonders for these children, and without such an improvement in general health no chemotherapy can give the maximum results.

In our experience gonococcal vulvovaginitis that is not sulphonamide-fast is best treated with sulphadiazine, as (in our hands) no toxic manifestations have been observed, such as may occur with the routine administration of sulphapyridine and (to a lesser degree) sulphathiazole. Resistant infections (and a relatively large proportion of such are sent for admission subsequent to failure as out-patients elsewhere) are notoriously difficult to treat. Encouraging results are being afforded by the local application of penicillin filtrate, but

restricted supplies do not yet allow of dogmatism. In the present state of our knowledge it would appear to be wisdom for all gonococcal vulvovaginitis infections to be considered as individuals and for no empirical treatment to be given as a routine measure. Furthermore, emphasis must be laid on the necessity for keeping these children under close observation for at least six months after the initial infection. Only thus will "relapses" be avoided and the danger of spread to other children in the home or the school be eliminated.—I am, etc.,

London, W.1. G. M. SANDES.

School Medical Service

SIR,—Dr. Gamlin's letter (Jan. 15, p. 90) would appear to be based upon a misunderstanding of the Education Bill. Were the position to be as he envisages it, then I fully agree that the results would be disastrous. The present routine cleanliness inspections are, however, carried out under the powers given by Section 122 of the Children's Act, 1908. This section it is not apparently proposed to repeal. It would appear to follow that the powers given by Section 52 of the Education Bill are supplementary to those already possessed and not in substitution therefor, which is a much more satisfactory position, though I doubt, personally, whether the proposed new powers will prove in practice to be of much use, since only cases of gross infestation are likely to come to the notice of the teachers.—I am, etc.,

A. C. TURNER,
Senior Medical Officer,
City of Leicester Education Committee.

Nightwear for the Troops

SIR,—I was amazed recently at the information given me by the wife of an A.C.2 that no provision of nightwear is made with the uniform. Further, an application for coupons to make good the deficiency was refused on the grounds that it was unnecessary. If this is the official policy in such ranks of the Services I am concerned, not as was my informant that those who refuse to lower their standards must provide nightwear at their own expense and that of their family's coupons, but that those with low standards receive no encouragement to raise them; rather they have the official seal set on them. One of the revelations, brought to light by evacuation, of the low standards of personal hygiene prevalent in the population was the absence of nightwear. In *Our Towns*, which so admirably surveys the many deficiencies found, one of the suggested remedies lies in adequate education. For many years, of course, health visitors and teachers have worked along these lines, but have been handicapped in part by ignorance that certain habits existed; and they in common with others were shocked when the closer contacts of evacuation showed them up. If every child spent a period at a residential school he would learn certain principles of hygiene by putting them into practice which he would never learn from blackboard instruction; but even if this were possible it would not be until the next generation that the full effect could be felt, whereas there is, in the masses of young adults now under discipline in the Services, a good field for practical health education.

Perhaps in some directions the opportunity is being used and the question of nightwear may seem relatively unimportant, but is it not true that such a simple process as changing the clothes helps to keep down the parasite population and thereby mitigate the disease and discomfort caused by parasites? In modern warfare there will inevitably be large numbers of men stationed permanently well behind the front line, and it is surely not necessary for the same standard to be applied to them. What do the medical officers to the Services think, or are they not consulted in such matters? I believe these remarks do not apply to the women's Services, so why the sex differentiation?—I am, etc.,

Leicester. JANET M. DONE.

Prostigmin in Delayed Period

SIR,—Dr. E. Friedmann, in the *Journal* of Jan. 1, suggests that prostigmin should be tried "in all cases of delayed period except those of endocrine origin and those in which pregnancy has been established." He classes as "unsuitable patients" those with "obvious endocrine disturbances, a history of long-standing amenorrhoea, or obvious menopausal symptoms," and relates that he has treated nine such patients "unsuccessfully."

What does he mean by "obvious endocrine disturbances" or "obvious menopausal symptoms"? The fact of the endocrine disturbance or menopausal symptoms being obvious or not depends only upon the powers of observation of the observer; the word "obvious" has nothing whatsoever to do with the condition of the patient and cannot describe it. What is obvious to one person may not be so to hundreds of others, and because a particular doctor does not notice a disturbance it does not mean that a disturbance is not present.

I would like to draw the attention of your readers to the fact that no good can result from Dr. Friedmann's recommendation that prostigmin be administered in "the treatment of delayed period," though they will possibly succeed in lulling their patients into a false sense of security if they act upon it.

He says that the drug should not be "tried where pregnancy has been established," yet in 30 out of his series pregnancy was the cause of the "delay"; pregnancy was "later discovered on clinical or biological investigations or the patients were known to be pregnant." Why does he recommend to others a procedure different from the one he himself has adopted? "Pregnancy," Dr. Friedmann says, "is not disturbed . . . prostigmin is safe . . . there is no tendency of the drug to interfere with the course of pregnancy." How is Dr. Friedmann to know that pregnancy has not been disturbed in some of his "successful cases"? It would seem beyond his power to establish a diagnosis of pregnancy in "a case of delayed period" without resorting to a "wait and see" policy or to help from laboratory tests.

He records that 24 of his cases were "poorly selected." Did he select them, and if so, why? He injects sterile water into 10 women: he observes, "No bleeding occurred." These cases must have been properly selected! For what reason does the injection of sterile water here prevent the menstrual flow? There is no reason why even a "delayed period" should not appear after such an injection, but in his cases the period obligingly waits for the "proper prostigmin course" which followed.

His experiments prove nothing and help nobody, and one wonders what ultimate good Dr. Freidmann hopes to have done by them. Should it not be the doctor's foremost endeavour first to decide whether the "delay" has occurred in the course of a harmless physiological process, or as a symptom of maladjustment leading to displacement and undue pressure on vital organs? Dr. Friedmann does not give any evidence that he even thought of assessing the influence of faulty functioning in his "diagnosis," which, therefore, was not a full and reliable diagnosis.

Is it not reasonable to consider and establish first the cause or causes, to ask first why a woman has a delayed period? Is it not misguided to produce the period at all costs, to "try prostigmin" and to proceed with "further clinical work to reveal the interesting effects of prostigmin in cases of dysmenorrhoea"? Dr. Friedmann will not find the means whereby to alter faulty functioning among the multitudinous variety of drugs on the market to-day, and though he may produce the period he does not put right the cause of the delay in such cases. And before proceeding with further useless experiments and recommending others to do likewise he would do well to become conversant with the work of F. Mathias Alexander, whose approach to all problems is first to establish beyond doubt the cause or causes, and who has provided a technique by means of which problems of faulty functioning can be solved.—I am, etc.,

London, W.1. DOROTHY S. RADCLIFFE DREW.

Heroin in Labour

SIR,—Dr. James Ross (Jan. 8, p. 59) expresses a view with regard to the safety of heroin in labour which is in conflict with pharmacological findings and recorded clinical experience in respect of this drug. That a drug stated to be five times more depressant to the respiratory centre than morphine (Dilling, *Materia Medica*, 16th ed.) should have this remarkable degree of safety in labour calls for some comment.

Favourable reference is made to heroin as an analgesic drug in labour in Williams's *Obstetrics* (7th ed.), where a dosage of 1/12 gr. is stated to have "no deleterious effect upon the child

providing the drug has not been given during the three hours preceding delivery." Clark in *Applied Pharmacology* (6th ed.) states that heroin "is in no way safer than morphine," and refers to heroin as being about 10 times as toxic as morphine. Heroin was introduced by Dreser in 1898. If Dr. Ross is correct in his estimate of its value in labour it is indeed remarkable that recognition of the value of heroin in this respect has been so long delayed.—I am, etc.,

Turriff, Aberdeenshire. A. T. FORBES.

Diet and Peptic Ulcer

SIR,—I am afraid that Dr. Maurice Webster has misunderstood Sir Arthur Hurst's meaning. One may disagree with Sir Arthur's speculative theories, as I do, but his factual observations cannot be assailed. I sympathize with Dr. Webster, but however hot his hyperchlorhydria may feel, a cold impersonal approach must remain the basis of all scientific investigation.

It is a practically daily observation, which is not sufficiently appreciated, that although hypochlorhydric patients are temporarily relieved by alkalis, their cure depends entirely on the administration of sufficient hydrochloric acid. "Heartburn" is a most deceptive symptom, and easily misleads the unaware practitioner, because his whole attention is focused on HCl as the cause of peptic ulcer. All authorities agree that HCl by itself cannot produce the ulcer. My own view is that hyperchlorhydria is the first manifestation of the preulcerative stage. All foods give a quantitative physiological secretory response. It will surprise many of your readers that gastric stimulation of water gives a positive response (Rehfuss *et al.*). The important point, however, is to discover what happens when the stomach is empty, for it is at that moment that the sufferer becomes aware of his symptoms. Whenever the stomach contracts it produces acid, but why should the stomach contract so violently when it is empty even a long time before any signs of chronic ulcer become apparent? The most likely explanation is that it reacts to some form of irritation, probably local. My explanation is that this occurs when the normal mechanics of the intestinal tract are upset—i.e., when iso-peristalsis is interfered with—by one cause or another. All the trouble begins from that moment. The implications of this thesis, however, are too complex to be dealt with in the short space of a letter.—I am, etc.,

London, W.1. J. JACQUES SPIRA.

Riboflavin in Hereditary Syphilis

SIR,—Recent articles in the *Journal* have shown the importance of the vitamin riboflavin as a therapeutic agent in inflammations of the eye and skin found in acne rosacea and also in cases resembling pellagra. In the keratitis associated with acne rosacea blood vessels and infiltrates are found in the superficial part of the cornea and, in aggravated cases, ulceration. The beneficial effect of riboflavin in rosacea inflammations of the eye has been known for several years; but it is not so widely known that this vitamin has also a curative effect in interstitial keratitis due to hereditary syphilis. Interstitial keratitis is characterized by the ingrowth of adventitious vessels into the substantia propria of the cornea as well as infiltration of cells. The following two cases show that riboflavin may be a therapeutic agent by itself.

Case 1.—A youth aged 18 attended the out-patient department of the Chelmsford Hospital with a keratitis accompanied by circumcorneal injection. Slit-lamp examination showed the lesion to be situated in the substantia propria of the cornea. No mention is made in the notes of corneal vessels. Wassermann and Kahn tests proved negative. Treatment by atropine and ascorbic acid failed to cause any improvement; sulphonamide tablets did not help. Carious teeth were extracted without avail. On Oct. 2 riboflavin (1 mg. t.d.s.) was substituted for the ascorbic acid. Compared with the former treatment it acted like a charm. On Oct. 16 the circumcorneal injection had disappeared as well as the corneal opacity. Shortly afterwards he joined the Army, having passed his medical tests.

Case 2.—A girl aged 13, sister of the patient in Case 1, attended the clinic on Jan. 20, 1943, both eyes having been affected by interstitial keratitis of several weeks' duration. The case was typical. There was photophobia and lacrimation. Both eyes showed the typical salmon patch due to corneal vascularization. Corneal opacity and iridocyclitis were present. Teeth were typically Hutchinsonian. Wassermann and Kahn tests were positive. The cause of the lesion in the brother's eye was now apparent. As he was several years older, the toxin had become more attenuated, the W.R. negative, and he showed a much milder corneal lesion. As riboflavin had caused such a marked effect in his case the same treatment was given to the sister. Notes record that on Feb. 20 the eyes were free from corneal injection. Later a complete course of novarsenobillon was given combined with riboflavin. Slit-lamp observation on Nov. 5, 1943, showed the cornea free from infiltrates and the corneal vessels represented by a few hair-like lines.

I ought to mention that in a case of acquired syphilis with deep vascularized keratitis and severe iridocyclitis in one eye treated by arsenical injections, riboflavin as an adjuvant did not appear to help. In the case of a young man with a vascularized keratitis of unknown origin, where the W.R. was negative and where syphilis could be excluded, riboflavin did not afford the expected improvement.

Will riboflavin prove a remedy in other manifestations of hereditary syphilis? In acne rosacea the ophthalmologist watches with interest the improvement which the administration of the vitamin effects in the skin of the face. In like manner the syphilologist may find a use for riboflavin in his department.—I am, etc.,

Chelmsford. S. G. CORNER.

Spontaneous Hypoglycaemia

SIR,—In your issue of Dec. 11 Dr. R. D. Lawrence expresses the view that spontaneous hypoglycaemia as an explanation for many minor indispositions has been overdone recently. Dr. Luntz, while supporting Dr. Lawrence, states that both hyper- and hypo-glycaemia are but "symptoms," but he rightly emphasizes that the nervous type of hypoglycaemia is a different phenomenon from that of hyperinsulinism, which term should be reserved for islet tumours of the pancreas. However, this statement, which suggests that the level of sugar in the blood is a "symptom," is one to which exception must be taken. Hypo- or hyper-glycaemia of itself can surely never be a symptom. The symptoms in neurotic patients to which Dr. Luntz refers are inevitably the reactions of the patients' nervous system to hypoglycaemia—if levels above 70 mg. per 100 c.cm. B.S. should be called by this name.

There is now evidence that individuals with cerebral dysrhythmia (about 10% general population and 26% of psychoneurotics) are particularly susceptible to lowering of the blood sugar, even to normal fasting levels. We have reported such a case, in which the electro-encephalogram (E.E.G.) was unstable below 100 mg. per 100 c.cm. yet stable above this level (*Lancet*, April 24, 1943). In these cases the symptomatology should not be regarded as purely hypoglycaemic, but as due to the increase of cerebral dysrhythmia which occurs. Our findings show that the normal E.E.G. does not exhibit instability at levels of blood sugar above 40 mg. per 100 c.cm., while dysrhythmics become unstable at higher levels. It has long been known that the onset of hypoglycaemic symptoms, as well as the onset of coma (Fraser, R., Maclay, W. S., Mann, S. A., *Quart. J. Med.*, 1938, **25**, 115), are not related to any one level of blood sugar, and vary from individual to individual. No doubt constitutional abnormalities of electrocortical stability are among the factors determining these differences.—We are, etc.,

DENIS HILL.
WILLIAM SARGANT.
MOLLIE HEPPENSTALL.

Chemistry of Vitamin Therapy

SIR,—The articles on vitamins in the *Journal* of Jan. 15 show, if I may say so with the deepest respect, a lack of appreciation of the chemistry and functions of these bodies. The addition of 25 mg. ascorbic acid to the diet of any person who already has enough can make no difference. If Prof. Davidson had first estimated the urinary excretion of vitamin C, and then confined his treatment to those whose excretions were below the minimum physiological standards of Harris, the recorded results would have been different. Similarly with the case of the compound vitamin capsule: what good can a jumble of vitamins do if the recipients are already taking a sufficient diet?

The weakness of both these experimental groups is that the individuals were all apparently well. My colleague and I offered for your acceptance some months ago a series of 600 analyses where the vitamin C excretion had been estimated

by Harris's three-hour technique, and which showed a significant difference between the apparently well and the sick; whereas the well averaged comfortably above Harris's line, the sick were consistently below it. So any addition of vitamin to the well cannot have any demonstrable effect. Vitamins are not fuel; they are catalysts whose presence is necessary for metabolism, but an excess of catalyst will not increase the magnitude of the chemical reaction.—I am, etc.,

Caernarvon. GRIFFITH EVANS.

The Rocking Stretcher

SIR,—Judging by the descriptions of rocking stretchers published in recent numbers of the *Journal*, there must be a competition to design some device or pivot to allow ordinary stretchers to be rocked. Mechanically these inventions are ingenious, and no doubt give great satisfaction to their inventors. Unfortunately, however, a rocking stretcher is intended for the resuscitation of the drowned or otherwise unconscious patient who is not breathing. I wonder how many of the inventors have considered the difficulty of safely securing an unconscious patient, who is limp and lifeless, to a rocking stretcher. To practise with a conscious subject is easy, but to fasten a toneless body on to a rocking stretcher by tying the ankles and wrists to the stretcher pole, as some inventors seem to consider satisfactory, is to court disaster. Serious damage to the limbs would be the least result, and I can imagine fractures of all types, even of the cervical spine.

I have recently had the experience of endeavouring to resuscitate an electrocuted girl of medium height and weight by means of a rocking stretcher. From this I can say that unless a proper brace, fitting between the patient's legs, dividing and fastening over the shoulders (similar to that provided on stretchers manufactured by Messrs. Siebe Gorman), is employed to keep the patient in position, an unconscious patient who is limp and flaccid would very quickly slide off the stretcher.

The inventors of rocking stretchers would be surprised to find how difficult it is to keep an unconscious patient safely fastened on to a rocking stretcher. Perhaps before any further methods of pivoting stretchers are invented they would do well to pause and consider this point.—I am, etc.,

Southwell, Notts. T. A. LLOYD DAVIES.

Cleansing Gas-contaminated Persons

SIR,—In the *Journal* of July 17, 1943 (p. 81), you published an article by Dr. Pollok Donald on a new method of gas training, together with a diagram illustrating a miniature gas-cleansing station. I entirely concur with Dr. Donald as to the apathy with which this subject is treated. If this apathy did not exist your correspondence columns would be full pointing out Dr. Donald's glaring mistake. It may be that he intended this to be so in order to gauge this apathy, as he says that his object is "not to instruct but to show how instruction may be given in a way acceptable to all concerned." His object is good but his diagram is dangerous.

If Dr. Donald is a gas instructor he should have read the official handbooks, in which case he would hardly have put his eye douche in the third compartment from the entrance to the station. This is most important, and the immediate treatment of blister gas in the eyes should be stressed to everyone in the country. The basis of all training is common sense, and this applies especially to anti-gas training. If anything falls in the eye obviously the first thing to do is to remove it mechanically. With a liquid, equally obviously the way to do this is to wash it out with large amounts of water. Applying common sense, the water should be warm to prevent spasm. As blister gas begins its foul work immediately it is only common sense to remove it at once. The penalties for delay are painfully obvious.

What happens in the gas-cleansing station in the diagram? The wretched patient arrives in a panic suspecting blister gas in the eye. Is his eye washed out at once? No. He has to go through endless formalities with labels, string bags, etc., and eventually is persuaded to have his outer clothing removed. He then goes through an air-lock, and entering another compartment is reduced to his birthday suit. Still clutching his valuables, which have been horribly in the way of undressing, he enters yet another compartment, where he joins the queue for the eye douche (singular). The proper place for eye douches (plural) is at the reception ("dirty") end of the gas-cleansing station, and liquid gas in the eyes *must be attended to at once*. It is advisable to have a store of anti-gas ointment in the "dirty" end. This is not marked on the diagram, but is doubtless not overlooked in practice. And this reminds me, every family should have a stock of anti-gas ointment (preferably No. 5) in good condition.—I am, etc.,

F. PITT PALMER,
Cmdr., R.N. (ret.)

Treatment of Coryza

SIR,—Adverting to Mr. Carew-Shaw's letter (Dec. 25, 1943, p. 830) regarding the treatment of coryza with ephedrine and argyrol in normal saline, I would like to point out that it appears to be quite beyond the ordinary pharmacist's facilities to produce a stable solution of these components. Undoubtedly many doctors will have been interested in Mr. Carew-Shaw's advice, and I feel that they should know that a stable solution is apparently only available in the form of a proprietary product. Unless this is made clear others will prescribe argyrol, ephedrine, and normal saline, as I did, only to find that a precipitate appears almost immediately, and that the resulting unstable solution causes congestion and crusting of the nose, as Mr. Carew-Shaw has observed.—I am, etc.,

London, W.1. A PINEY.

Fact and Fancy in Poliomyelitis

SIR,—My attention has been drawn to the leading article in the *Journal* for July 31, 1943. This is in part a review of the book published by the Bruce Publishing Company, St. Paul, Minnesota, and presented by Dr. J. F. Pohl and myself, with the foreword written by Dr. Frank Ober, professor of orthopaedic surgery, Harvard University, and commentary by Dr. Miland Knapp, professor of physical therapy, University of Minnesota.

At the onset you state: "It is permisible to regret that Sister Kenny was not content to remain an outstanding practitioner of her particular art, and that she succumbed to the temptation to rationalize her methods by providing a scientific basis for them." I may here emphatically emphasize the fact that there was no one on earth more content to pursue the even tenor of my way than I, right from the onset in my early Australian days, and that all my activities have been by special request. The public presentation of my work in Australia was made by special request. My visit to the United States was arranged by my own Government and a group of medical men in Australia, who wrote to the senior orthopaedic surgeon of the world-famous Mayo Clinic. My association with the University of Minnesota was made by special request. My association with the University of New York was by special request. My position as honorary director of the Elizabeth Kenny Institute was accepted by special request.

You say that I have formulated an entirely new concept of the disease. This is also the opinion of all medical observers. It was first published by Dr. Wallace Cole, professor of orthopaedic surgery, and Dr. Miland Knapp, professor of physical therapy, University of Minnesota, in the *Journal of the American Medical Association*, June 7, 1941. In this paper they state that the concept of the disease is entirely the opposite of that previously recognized, and this new concept had been proven in 100% of cases. This is also the opinion of Dr. Frank Krusen, professor of physical therapy, Mayo Clinic, published in the paper presented in the *Staff Bulletin*, Aug., 1942. In this paper Dr. Krusen admits that he thought I was unbalanced when I arrived in the United States and presented my ideas, but added that he admitted "she has presented to us an entirely new concept of the disease anterior poliomyelitis and a satisfactory treatment for this concept." Before my arrival in the United States the percentage of recoveries according to statistics published in the *Journal of Bone and Joint Surgery*, Oct., 1941, was 13. Since my arrival in the United States the recoveries are 88%. I have also been requested to assist in preparing a book on the muscle physiology and function. This is now in operation.

I am quite sure that if your Association could see its way clear to send a suitable physician to Minneapolis we would be in a position to present sufficient evidence to prove that the old concept of the symptoms presenting themselves in this disease was incorrect. I am also happy to say that I could prove to him that there is a certain condition existing in this disease which indicates that portion of the body in which the disease is likely to attack, and by the prompt administration of treatment the progress of the disease can be arrested in a great many instances. It has also been acknowledged that the physical therapy procedures introduced by me have proved most advantageous in other types of neuromuscular injuries and war wounds.

I quite understand your criticism. As a matter of fact it is extremely mild in comparison with the criticism I first met with in this country by those who now strongly support my work. Therefore I would very much appreciate the publication of this explanation.—I am, etc.,

Minneapolis. E. KENNY.

Photomicrography with Ordinary Cameras

SIR,—I was much interested in the article on photomicrography with ordinary cameras by Dr. W. N. Leak (Dec. 18, p. 787), and would like to point out that colour photomicrographs and the method of taking them with ordinary cameras were demonstrated by me at the Dermatological Section meeting of the Royal Society of Medicine held in London on March 19, 1942, when I showed colour photographs and photomicrographs of sections of two rare skin diseases.

The method of taking colour photomicrographs without the use of any special apparatus is as follows: "The section is placed and focused under the microscope in the ordinary way, then the microscope with the slide in position is tilted into the horizontal position and the reflector adjusted to give good illumination. The miniature camera, a Zeiss Ikon, is brought up to the ocular of the microscope in the same horizontal plane till it just touches firmly. All extraneous light is excluded by means of some suitable covering—e.g., a piece of dark cloth wrapped round the junction. Film used was Kodachrome A (for artificial light). One photoflood light was used and reflected as described above. The camera lens was set for infinity, the aperture used was f/3.5, and the exposure was one-fifth of a second. One-tenth of a second was also tried, under the same conditions, but was not sufficient. I hope to make further trials of this simple method of taking coloured photomicrographs as it does not require any special apparatus" (vide *Proc. roy Soc. Med.*, 1942, **35**, No. 4). I might add that the colour photomicrographs referred to above were shown in the scientific and technical section of the 1943 Annual Exhibition of the Royal Photographic Society of Great Britain in London.

Colour photomicrographs are very much more interesting to look at, and lend themselves as an ideal means of projection by lantern for demonstration at meetings, etc.—I am, etc.,

Leicester. F. A. E. SILCOCK.

Emulsions

SIR,—May I suggest that there is another aspect of emulsions than that mentioned by you on Dec. 18 at page 803, one which is really of great importance to practical medicine, though it is seldom considered. The work is very old. So far as I remember, Pickering, in the last century, showed that a solution of phenol in water might have little disinfecting power; but if an emulsion of some inert oil, or very fine inert solid particles, were added the bactericidal power was increased. This was followed by many others, and, so far as disinfectants go, gave rise to those with "high coefficient."

There is a boundary between a drop of water in oil or of oil in water. This boundary is due to electric charges—molecular attraction, if that term is preferred—which cause the drops to be round, and which also include surface tension phenomena. The less the diameter of the drop the greater the force per unit area; so that when the diameter is extremely small the force is comparatively very great. The explanation of Pickering's work would be that the molecules of phenol are attracted to the interface, so that the concentration per minute area is very great; whereas the concentration per c.cm., say, is comparatively small. The bacteria also have charges on their boundaries, and are also attracted to that interface (oil–water), so that they are exposed to the comparatively strong concentration of phenol there.

This subject is a very large one and concerns many things in medicine, such as the function of the blood corpuscles, tissue cells, and so on. The odd thing is that, although it is mentioned in textbooks on physiology, it seems to be completely ignored in practical medicine; yet it is of much importance in that sphere.

Dealing with dermatology, I should say that the subdivision of the remedial agent increases its "potency," because the charges at the interfaces will react with charges on the prickle cells, or on the prickles themselves; so that in the first case there will be stronger reaction between the agent and the cells; in the second, the agent will be able to pass more readily between the prickles and affect the cells underneath. The result might be a variation in the outermost cells or in those below them. I know of no proof that the prickle cells have a charge, but they are chitinous, and there is definite proof that emulsions wet chitin more readily than water—the nail, for example.

The "value" of an emulsifier will depend on its ability to reduce the oil (to use popular terms and not phase-theory terms) to extremely minute drops; and that is why some of the modern emulsifiers are more useful. So the "value" can be estimated by such determinations as the surface tension, number of globules, opacity, and so on. The detergent effect of soaps is due to this property—it is really a fine emulsion. A solvent will wash away a greasy mark on the skin by dissolving the grease, and often substances from the cells of the skin. A soap acts by emulsifying the grease so that it is washed away with the dirt by plain water. If the soap is alkaline it will also affect the cells; if it is superfatted it will tend to supply some fat to the cells (it also annuls the alkalinity of the soap due to dilution).

All this business of charges, etc., is very intricate and requires a good knowledge of mathematics. But it is not necessary for the practitioner to know that: only that he shall understand the results and be able to apply them in practice. So I suggest that there is really great scope for some medical man who is lucky enough to know sufficient maths. to write up an account of it—as to how, when, and where it *may* affect practice. For instance, it must be concerned in the problem of feeding and digestion of milks; for the globules of fat vary in diameter and the protein adsorbed to the globules varies. One peculiarity concerns the dermatologist—namely, that there is evidently some practical difference between solutions, emulsions, and ointments. One can compare the effect of a "blob" of ointment containing, say, 30% of calomel with a solution of mercury biniodide 1 in 1,000 or more dilute as preventive against spirochaetes.—I am, etc.,

Kingston-on-Thames. W. F. COOPER.

Malaria among Troops in W. Africa

SIR,—Your leading article (Jan. 15, p. 84) starts with a most misleading sentence. Your writer can have little personal knowledge of the place or he would remember month after month of perpetual Turkish baths, with no possibility of a change to a better climate till his next leave was due. He would remember the thirst produced by this heat and the boredom after sundown, and would probably think that he had consumed more alcohol than was good for any man. Could he forget yellow fever? Malaria would have bothered him little more than influenza in an English winter. Malaria is a mere fraction of West African unhealthiness.—I am, etc.,

O. G. WILDE.

*** By "unhealthiness" in inverted commas was meant a reputation for unhealthiness. It was not intended to imply that there were not other causes of ill-health in the climate of the West Coast of Africa and other tropical countries. The statement was an expression of the writer's own experience of life in many tropical climates, and is, he believes, generally speaking, true. Probably it is the heavy mortality from blackwater fever—the result of malaria—which has usually been responsible for the gaining of such reputations. Yellow fever was in the past, again as a result of mortality, perhaps in the New World an even greater originator of evil reputations, but at the present time things have greatly changed.—ED., *B.M.J.*

Obituary

MAITLAND RADFORD, M.D., D.P.H.

Dr. Maitland Radford, whose death at the age of 59 occurred on Jan. 19, was one of those excellent medical officers of health who are able to achieve the most satisfactory administrative results with the minimum of officiousness and dissension. This ability was due partly to his wide knowledge of public health and his very alert and progressive mind; partly to his tact, arising from a natural sensitiveness to the feelings and opinions of others; partly to his charm, which was assisted not a little by his keen but kindly sense of humour and ready wit; and partly to the conscientious and energetic way with which he pursued his objects without the slightest regard to personal ambition. In spite of his urbane and generous manner, and though the overriding of other people's opinions was so far from being a pleasure to him that it was often an obvious embarrassment, nothing would induce him to do or say anything which conflicted with what he thought was right. In respect of his principles he was adamant and fearless.

Maitland Radford was the son of Ernest Radford, a barrister who lived at Hampstead, and who was also a progressive thinker and a friend of Shaw, Wells, William Morris, and Bradlaugh at a time when many of their ideas, which are now commonly accepted, were regarded with suspicion and even with hostility. There is little doubt that this background of controversy in the cause of rational thought and action had a profound influence on Radford's outlook and character. He was educated at Abbottsholme School, Derbyshire, and completed his medical studies at University College Hospital, where he qualified in 1912. In 1914 he took his M.B., B.S.Lond., and during the war joined the R.A.M.C. as a temporary medical officer and was posted to No. 3 General Hospital in France. Later he returned to complete his study of public health at the North-Eastern Fever Hospital and Queen Mary's Hospital, Carshalton, and during this period he married Muriel Ann Lloyd, also a doctor, and the daughter of Dr. Henry Lloyd of St. Asaph, Wales. In 1920 he took the M.D. in State Medicine and D.P.H. London, and in March, 1921, was appointed assistant M.O.H. to the borough of Shoreditch, where, after promotion to deputy, he became medical officer of health in 1927. He is still remembered in that borough for his efficiency, his easy and courteous manner, and especially for the great interest he showed and the considerable improvements he made in the maternity and child welfare scheme.

But though Dr. Radford devoted much energy to his immediate work he was not a man to allow the local trees to obscure his vision of the public health wood—or, as he might have said, the public health jungle—as a whole. He took a deep interest in general problems of public health, particularly in housing and health propaganda. He was a founder-member of the Central Council for Health Education and had much to do with the inception of that important body. For many years he was its honorary treasurer, and at the time of his death held the position of vice-chairman. He was also an active member of the Society of Medical Officers of Health, which he joined in 1920 and in which he was president of the M. & C. W. Group in 1931–2 and president of the Metropolitan Branch in 1940–1. In 1934 he became M.O.H. of the borough of St. Pancras, where his many good qualities were speedily recognized. He was chairman of the St. Pancras Division of the B.M.A. in 1936 and again in 1943. In 1938 he was a vice-president of the Section of Public Health and Hygiene.

The work of an M.O.H. is not often of a very dramatic kind, nor do the general public for whom he works often appreciate or even realize what is being done for them by a capable man in his position. It is, however, no exaggeration to say that there is no member of his staff or of the borough council who will not regret the loss of his leadership and that he will survive in the minds of many, not only as a memory, but as a permanent influence on their thoughts and actions.

C. H. writes: Radford was, in the best sense of the word, an intellectual. Essentially humble, his scintillating mind leapt from idea to idea, flashing a light just long enough to make clear his point before the beam passed with amazing swiftness to yet another idea. It is surprising that he found satisfaction in an administrative life. One could picture him as a university don, as a teacher, for he was above all a thinker. In the Central Council for Health Education he was a fountain of ideas, prodding the conventional, flicking the timorous with a wit which was as kind as it was charming. His interest was in ideas and principles, and he could be passionate in their defence. A good treasurer, though he knew and cared nothing for accounts; an efficient vice-chairman, though procedure left him cold, he would have been chairman, and a great chairman, in a few months' time. He leaves a gap which cannot be filled.

F. WILFRED WILLWAY, M.D., M.S., F.R.C.S.

We regret to announce the death on Jan. 6 at the early age of 36 of Mr. Francis Wilfred Willway, temporary assistant surgeon to the Bristol Royal Hospital and a former Hunterian Professor of the Royal College of Surgeons. He was the son of the late Dr. Frederick Willway of Streatham, and studied medicine at King's College Hospital, whence he qualified in 1930 after graduating B.Sc.Lond. with honours in 1928. In successive years he took the M.B., B.S., the F.R.C.S., the M.S., and the M.D., and after working as surgical registrar and casualty officer at King's he became in turn senior house-surgeon at the Royal East Sussex Hospital, resident surgical officer at the Royal Manchester Children's Hospital, and assistant surgeon to the West End Hospital for Nervous Diseases.

Prof. GEOFFREY JEFFERSON writes:

British neurosurgeons deplore the death of Wilfred Willway. It is unlikely that any of us will again see such a combination of debonair heroism and sound practical utility as his life showed, not paralleled even by Hans Zinsser, who had a shorter battle to fight and was an older man. Few people realize that death is close upon them, and none that I have known have treated the knowledge with Willway's good-natured disdain. Its gradual inevitability over a period of years failed to impair either his cheerfulness or his rooted determination to utilize to the full his many gifts in the service of his fellow men. He often spoke of his impending death and his determination that the knowledge should not deflect him from his duty. This was particularly evident at the height of the air attacks on Bristol, where he overdrove his ailing body. What was extraordinary about Willway was that he expressed no bitterness at the shortness of his days, no complaints that he would never see the fruition of his plans and dreams. None the less he accomplished a great deal. Some of his work cut into new ground (leucotomy), and he deserves great credit for his enterprise there, but he did also a great deal of solid work on more conventional lines. He had a brilliant, a mobile, intelligence and a great disregard for the sanctity of the opinion of his elders, from which all edge was removed by the knowledge that all that he had to say he said at once. And how entertaining he could be! We neurosurgeons cannot afford these losses from our ranks that the last three months have brought. Willway and Eden were a notable pair, and however time chances to fill the gaps those brave personalities are for ever lost.

A surgical colleague writes:

Wilfred Willway was one of the most brilliant students of King's College Hospital. He had that type of brain which makes its possessor able to play four games of chess and conduct a conversation at the same time. He never failed in any examination, took the F.R.C.S.Eng. and M.S.Lond. at an early age, and then the M.D. "just for fun," as he explained. He was a great teacher, whose students admired and loved him as did all his friends. As a man he was full of kindness and humility and yet charged with enthusiasm for his work and the welfare of his patients, students, and friends. Coming to Bristol from London, Willway entered fully into the teaching and professional work of the Bristol Hospitals and Medical School. He was a pillar of strength to the hospitals' staff, who, because of his sound organizing powers, gave him charge of the war emergency arrangements at the Royal Infirmary. Willway was Bristol's first neurosurgeon, and he was rapidly achieving a wide reputation for his work at the infirmary and the Burden Neurological Institute. He was particularly interested in the surgical treatment of mental disease and had performed more leucotomies than any other surgeon in this country. His papers on this subject in collaboration with neuropsychiatrists are highly important. For years he made a heroic struggle against the ravages of a remorseless chronic malady which from time to time laid him low, but each time he came up smiling again and would laughingly chat about the prognosis. His courage was admirable, and in the course of a long day's operating it is reported that on one occasion he fainted twice. His tall spare frame, high forehead, and rather prematurely bald head, with merry eyes behind his large glasses, were the introduction to a personality which was widely loved and will be sadly missed. He was on a plane above most people, and Bristol is a poorer place for his departure.

G. H. PEARCE, M.D., M.R.C.P., D.P.H.

Dr. George Herbert Pearce, who died on Jan. 6, had been a member of the B.M.A. for 45 years. He was vice-president of the Section of Public Medicine and Industrial Diseases at the Bradford Annual Meeting in 1924, was chairman of the Dewsbury Division in 1931–2 and represented it at nine Annual Meetings, and became a member of the Insurance Acts Committee in 1936. He took the Scottish triple qualification in 1897, the D.P.H. of Cambridge in 1905, the M.D. of Durham in 1914, and the M.R.C.P. in 1935. His successor, Dr. W. J. Frain, sends the following appreciation :

The death of Dr. G. H. Pearce removes one of the most prominent personalities in public health during this century. He had been M.O.H. for Batley for almost 30 years before his retirement 5 years ago. His great gifts of personality and ability soon began to be felt in his profession and his influence extended far beyond the boundaries of Batley. He worked hard to improve the status of the medical officers of health in the country, and those of us who belong to the younger school of M.Os.H. owe a great debt to his untiring efforts on our behalf. He was an outstanding figure in the Society of Medical Officers of Health, serving as its president in 1932. He was well known at the Ministry of Health and was a friend of nearly all its leading medical officers. At Batley he produced a public health department which was probably second to none in the country. He was a gifted administrator, and as he was also a barrister-at-law his legal knowledge proved a great asset. He approached his problems fearlessly, the health and welfare of the people of Batley being his first concern, and with a stern sense of duty he attacked all obstacles. From his staff he demanded first-class efficiency. He was deeply touched when the Batley councillors combined to make him a presentation on his retirement, for he had the erroneous idea that he was not a popular man. He always contended that if an M.O.H. did his duty he could not expect to be popular with the community. Those of us who mix among the people of Batley know how much Dr. Pearce was respected for this quality of devotion to duty. During his retirement he did part-time service for the Ministry of Pensions at Leeds, a work which gave him great joy and satisfaction. He attended regularly the meetings of the Yorkshire Branch of the Society of Medical Officers of Health and was a very valued member, owing to his rich experience in procedure. He was ever ready to help the younger members of the profession with his wide knowledge and sane counsel. His fine Victorian courtesy of manner made him a charming companion socially, and this was particularly evident in his home.

PROF. ALFRED VOGT, M.D.

Prof. Alfred Vogt, the famous ophthalmic surgeon and director of the University Eye Clinic, died at Zurich on Dec. 10, after a long illness, at the age of 64. He had resigned his chair at the university earlier in the year on account of ill-health.

Alfred Vogt was born in Switzerland on Oct. 31, 1879, and after studying at Aarau Gymnasium he went to the Universities of Basle and Zurich, graduating M.D. in 1902. There followed a series of house appointments, at the end of which, in 1909, he became chief resident physician at Aarau Hospital. There he was to spend the next nine years till his appointment as professor extraordinary at Basle, where in 1920 he was elected to the chair of ophthalmic surgery at the university. He was immediately put in charge of the research department, where he devoted himself with characteristic energy to the problems of senile cataract. He edited at this time Graefe-Semisch's popular manual, *The Pathology of the Lenticular System*. In 1919 he perfected the slit-lamp microscope, which is a binocular mechanism based on an optical system, for the focal illumination of the eye, as well as being responsible for the introduction of novel operative procedures into ophthalmic surgery. His celebrated *Text-book and Atlas of the Slit-Lamp Microscopy of the Living Eye* was published by Springer from 1921 to 1931. To facilitate the detailed examination of the retina he introduced infra-red rays into ophthalmology, and, in order to study the true nature of snowflake and glassblowers' cataracts, he carried out experimental studies on the effects of various rays on the eyes. His subsequent researches, prosecuted with his usual thoroughness and enthusiasm, dealt with the healing of detached retinae, with his discovery of glaucoma of the lens capsule, and with various forms of cataracts. In his investigations on the optical constants of the eye and on the crystalline lens he approached Helmholtz in versatility. In 1923 he was invited to assume the chair of ophthalmology at Zurich and the direction of the University Eye Clinic. It was from Zurich then that he achieved the widest international celebrity, though his basic work was done during his Basle years. He brought to the problems of ophthalmic surgery extensive knowledge of biology, internal medicine, and physics. As often happens to historic and popular figures, public attention has been focused on his skilful operations, his brilliant technique, and the long pilgrimage of distinguished men who flocked to his clinic; but his fundamental contribution to medical science has been overlooked. However, in December, 1942, the Swedish Medical Society of Stockholm awarded him its most-coveted Gullstrand Medal, presented every ten years to the most distinguished ophthalmologists. We mourn with our Swiss colleagues the untimely death of one whose name ranks among the greatest of surgeons and whose work will long remain the embodiment of all that is best and noble in the traditional culture of Europe.

R. E. G. A.

Dr. RICHARD BEVAN LLEWELLYN, who gave long and faithful service as assistant medical officer under the Glamorgan County Council, died at his home at Llanishen, near Cardiff, on Dec. 1, aged 56. Fifth son of the late Alderman William Llewellyn, a former chairman of the Glamorgan County Council, he studied medicine at the University of Edinburgh and graduated M.B., Ch.B. in 1913, and later in the same year was appointed to the post he held until his death. During the last war Dr. Llewellyn served as a temporary captain in the R.A.M.C. in the Near East and Far East, and he joined the B.M.A. in 1917. He is survived by his wife.

Worcester and district has lost its leading ophthalmologist by the death after a short illness of Mr. BERNARD HAMILTON ST.CLAIR ROBERTS. He died at his residence near Worcester on Dec. 19. He had suffered from an infantile paralysis since childhood. St.Clair Roberts was the son of the late J. W. Roberts, L.D.S., of Dudley. He studied medicine at Mason's College, University of Birmingham, and was one of the senior prosectors in anatomy. He qualified M.R.C.S., L.R.C.P. in 1900 and was house-surgeon at Wolverhampton Eye Infirmary, senior house-surgeon at the West Bromwich District Hospital, and for three years house-surgeon at the Birmingham and Midland Eye Hospital. He was early appointed ophthalmic surgeon to the Guest Hospital, Dudley, and held that office until 1940, when he was appointed consulting ophthalmic surgeon. He came to Worcester in 1908 and was the first consultant to settle in the district. In 1911 he was appointed surgeon to the Worcester Eye Hospital, and he took a very active part in the work of that hospital right up to the time of his illness, only four days before his death. He was very largely responsible for keeping the hospital up to date, and was instrumental in acquiring new premises, when the work done made that an urgent necessity. He was beloved by all his patients, both rich and poor, and he was a true friend to them all. His opinion was widely sought by the medical practitioners in the district. He was punctilious in the performance of his duties at the hospitals which he served. He was ophthalmic surgeon to many of the education committees in the County of Worcester, and he had established an orthoptic clinic at the Worcester Eye Hospital. St.Clair Roberts had a most generous disposition and had no enemies. He had been a member of the B.M.A. for about 40 years, and in 1931–3 held office as president of the Worcester and Hereford Branch. He was a very active member of the Oxford Ophthalmological Congress and never missed a meeting; he was also a member of the Ophthalmological Society of the United Kingdom and attended meetings of other scientific societies. A memorial service held at the Worcester Cathedral on Dec. 23 was attended by many of his medical colleagues, friends, and patients. The address was given by the Dean of Worcester, who is president of the Worcester Eye Hospital. Mr. St.Clair Roberts leaves a widow and one son, who is studying medicine at Magdalen College, Oxford.—H. N. C.

Dr. FLORENCE SCOTT KIRK died suddenly on Dec. 22, while on her way to the Jessop Hospital for Women, Sheffield, to carry on her duties as pathologist. The daughter of Dr. Robert Kirk of Glasgow, Miss Kirk was born on March 8, 1889. She was educated at the Glasgow High School for Girls and at Glasgow University, where she graduated M.B., Ch.B. in 1915. She held resident appointments at the Royal Maternity Hospital, Glasgow, and the Royal Infirmary, Oldham, and in 1917 became a civilian medical practitioner attached to the R.A.M.C. In 1918 she was transferred to the 1st Eastern General Hospital, Cambridge, as pathologist, and when that hospital was closed in 1919 she was transferred to the Central Military Laboratory Medical Schools, Cambridge, as assistant pathologist. She left the Army service in April, 1920, and became junior assistant in the Pathological Institute of the Royal Infirmary, Glasgow, under Prof. Teacher. Two years' work there was followed by her appointment as assistant pathologist to the Victoria Infirmary, Glasgow, where she worked for three years, and in 1925 was then appointed assistant pathologist to Sheffield University and the Jessop Hospital for Women. In 1923 she obtained the Cambridge D.P.H. For the past 18 years Miss Kirk has worked at the Jessop Hospital, and with the

passing of the years she was constantly enlarging her knowledge of gynaecological pathology, keeping pace with the many advances that have taken place during this time. Her work was conscientiously carried out. Her opinion on specimens submitted to her was carefully considered before being expressed. It was always sound, and was greatly valued by the hospital staff. There is no doubt that the fine work she has done for so many years was a most important factor in the success of the hospital practice. Apart from her medical work, Miss Kirk was greatly interested in social problems, and devoted much time and thought to them.

Dr. ALFRED JOSEPH PICKWORTH, who died in retirement on Dec. 23, aged 86, had practised for over half a century in the Lakenheath district of Suffolk. He came of a medical family, for his grandfather, two uncles, and a son have also been members of the profession. He was born in Lincolnshire and went to school at Bath, and studied medicine at Liverpool, qualifying as L.R.C.P.Ed. and L.R.F.P.S.Glas. in 1880. Four years later he went to Lakenheath, where he became not only the medical attendant and adviser of the people of his large practice but also took duty as a Methodist minister, and his medical and religious work were closely intertwined. Dr. Pickworth was prominent in local public life, a J.P. for Suffolk, and in 1935 the County Council advanced him from the position of a councillor to that of an alderman. He was a governor of Addenbrooke's Hospital, Cambridge, and for years attended the Mildenhall Cottage Hospital. On his retirement from medical practice a large gathering assembled to do him honour and make a presentation to him and Mrs. Pickworth. He was a member of the B.M.A. from 1881 to 1930, but took no part in medical politics.

We regret to announce that Dr. JOHN CHARLES WILLIAM GRAHAM, a well-known Cambridge practitioner, died on Jan. 1, aged 72. From Brighton College he went up to Trinity College, Cambridge, where he distinguished himself as a swimmer and member of the University water-polo team. Having taken the B.A. in 1893, he went to St. Thomas's Hospital for clinical study, qualifying in 1899, after which he returned to Cambridge as house-surgeon at Addenbrooke's Hospital. He worked for a time at High Wycombe and then returned again to Cambridge and set up practice, taking his M.D. in 1909. He was chiefly interested in ophthalmology and acted as clinical assistant at Addenbrooke's for many years, and also as consulting ophthalmic surgeon to the Huntingdon County Hospital. During the last war he served on the staff of the 1st Eastern General Hospital, retiring in 1919 with the rank of major, R.A.M.C.(T.F.). Dr. Graham was a member of the B.M.A. for over 40 years and took an active interest in its affairs. He represented the Cambs and Hunts Branch at a number of Annual Meetings.

By the death of Lieut.-Col. A. E. J. LISTER I have lost a life-long friend, writes Mr. J. Cole Marshall. We were at Dean Close School, Cheltenham, together, and then we both went on to St. Bartholomew's Hospital, where Lister passed all his examinations with ease, also taking the Brackenbury Medal—the blue ribbon of the surgical scholarships. Lister was also prosector at the Royal College of Surgeons. After graduating M.B. and B.S.Lond. he took his final Fellowship, passing this examination under age. He then decided to enter the Indian Medical Service, obtaining first place in the entrance examination and passing out first, being presented with the bronze medal for efficiency. Soon after starting his career in India Lister was attached to the 27th Punjabis, and at the Battle of Jid Boli he rescued a wounded officer under fire when the stretcher-bearers had bolted: he not only saved the officer's life but stopped his arm being amputated, thus allowing him to continue his career and eventually to become a general in the Indian Army. In this campaign Lister caught a fever which undermined his health for the rest of his career. I would especially like to dwell on his ophthalmic work. On returning from the campaign he was attached to the staff of H.E. the Commander-in-Chief of the Indian Army, Sir C. MacCreagh, and remained with him until 1913. During his leaves he studied ophthalmology in Vienna under Prof. Fuchs, and also worked at Zurich and Paris; being very proficient in languages he naturally became well known in those universities. During these years he worked with Lieut.-Col. Henry Smith at Jullundur and Amritsar; he wrote an appendix to Colonel Smith's book on treatment of cataract by the intracapsular method, and also analysed the cases of vitreous loss in this operation. He also wrote a paper on conjunctival flaps and their use in the intracapsular operation. Lister was a good and quick operator and had a great experience in cataract. In 1913 he inaugurated the ophthalmic department of King George's Hospital at Lucknow. He first was appointed professor of physiology, and then, in recognition of the way he developed the ophthalmic department, was made first professor of ophthalmology. In 1920, owing to the great increase of eye work, a new wing was in course of construction, but though Lister had hoped to return to India, in 1920 he had to retire from the I.M.S. because of ill-health. His ophthalmic work in India was greatly appreciated; he was appointed ophthalmic surgeon to two Viceroys and was consulted by many of the leading Indian Princes and high officials. On his return to England Lister started consulting practice and was appointed assistant surgeon to the Western Ophthalmic Hospital, but in 1923, owing to ill-health, he went to Bristol, where he was attached to the Bristol Eye Dispensary, reorganizing the work of the hospital. He worked at Bristol for many years and had a considerable consulting practice. In 1933 he retired from his hospital work because of poor health, but still took a great interest in the institution. Lister was a wonderfully kind-hearted man, and well loved by all who came in touch with him, both in his private and hospital work. Considering his ill-health it was marvellous how he carried on for so many years. In ophthalmic literature his name was well known, as he contributed to the Ophthalmic Section of the *Medical Annual* from 1922 to 1930. Lieut.-Col. Henry Smith, I.M.S., writes: After Col. A. E. J. Lister's return from sick leave from Somaliland fever early in his career he was posted to Jullundur, where I first became acquainted with him. From cantonment to my hospital at Jullundur city was 3 to 4 miles. While there I invited him to come daily to my hospital and work with me in his spare time, which he took full advantage of. The variety of clinical material was unsurpassed in quantity and quality, with only one boss—myself. How he enjoyed the hard daily steady work! A true and loyal friend, an honest and courageous man, and the finest Christian I ever met. I mourn his loss.

The Services

The *London Gazette* has announced the appointment as M.B.E. (Military Division) of Flying Officer Robert Dunlop, R.A.F.V.R., and the awards of the George Medal to Capt. C. E. L. Allen, S.A.A.F., and Flying Officer G. H. Dhenin, R.A.F.V.R., and the B.E.M. (Military Division) to Corporals C. V. Burton, R.A.F.V.R., and W. J. Lush. The announcements read as follows:

F/O ROBERT DUNLOP, R.A.F.V.R., and Cpl. C. V. BURTON.

One afternoon in July, 1943, an aircraft crashed on landing and caught fire. The pilot and a passenger, who were the only occupants, were both thrown out of the cockpit and trapped beneath the wreckage of a wing. Cpl. Burton, assisted by two other airmen, attempted to rescue the occupants, but all were driven back by the heat. Undeterred, Burton made another attempt alone, and this time succeeded in partially lifting the burning wreckage of the wing and he then dragged the pilot clear. F/O Dunlop, a medical officer, had arrived at the scene of the accident, and on being informed that the passenger was still under the wreckage, attempted to release him. While this was being done an explosion occurred, but F/O Dunlop continued his rescue efforts and dragged the passenger clear of the aircraft. This officer and airman displayed considerable bravery.

Capt. COLIN ERNEST LEWER ALLEN, S.A.A.F.

One night in July, 1943, an aircraft, with a crew of four, collided with a stationary aircraft and two trucks when taking off. The bomber, which was carrying six 250-lb. bombs, burst into flames. Capt. Allen, the squadron medical officer, after instructing an orderly to bring a near-by ambulance to the aircraft, proceeded to the scene of the accident. Before he was able to get to the wreckage he saw one member of the bomber's crew stumble away to safety. Then the starboard petrol tank exploded. On reaching the aircraft Capt. Allen saw the pilot, who was injured and in a dazed condition, in the nose of the aircraft endeavouring to locate the observer. After removing the pilot Capt. Allen groped in the nose of the bomber, but was unable to find the observer. He continued his search around and over the port wing, which was well ablaze, in an endeavour to find the two remaining members of the crew. Fully aware of the danger from the possible explosion of the bombs, Capt. Allen remained in the vicinity, and finally, with an assistant, he removed the body of the gunner, which was found lying partly under the wing beside the blazing fuselage. Shortly afterwards three of the bombs exploded. Capt. Allen displayed great courage and devotion to duty.

F/O GEOFFREY HOWARD DHENIN, R.A.F.V.R., and Cpl. W. J. LUSH.

One night in October, 1943, an aircraft, which had sustained damage during an attack against Hanover, crashed near an airfield. The aircraft disintegrated on impact and immediately burst into flames. The rear gunner was injured and trapped in his crushed turret, being pinned down by the remains of the tail unit and the rear of the fuselage. A high-explosive bomb was in the blazing wreckage some 10 yards away from the gunner. F/O Dhenin, the station medical officer, and Cpl. Lush, a gunner, hastened to the scene of the accident. Although fully aware that the heat might cause the bomb to detonate at any moment F/O Dhenin worked for over half an hour to relieve the injured airman's pain and, assisted by Cpl. Lush, endeavoured to release him. Their efforts to extricate the gunner were, however, unavailing. A mobile crane was brought to the scene and the mass of wreckage was lifted clear of the ground. Displaying complete disregard for his own safety, F/O Dhenin then crawled under the wreckage and released the trapped airman,

thereby enabling other helpers to drag him to safety. F/O Dhenin and Cpl. Lush showed fine courage and determination in circumstances of great danger.

The following have been mentioned in dispatches in recognition of gallant and distinguished services in the Middle East:

Brig. (Temp.) J. Walker, C.B.E., M.C.; Col. H. J. A. Longmore, late R.A.M.C., Brig. (acting) J. M. Macfie, O.B.E., M.C., Brig. (local) D. McAlpine; Cols. (Temp.) D. Mackie, M.C., T.D., D. McVicker, M.C., F. R. H. Mollan, O.B.E., M.C., I. M. Pirrie, M.C., and W. Russell, M.C.; Cols. (acting) C. Donald, C. E. Eccles, and R. Marnham; Majors (Temp. Lieut.-Cols.) I. Aird, C. Bainbridge, A. MacC. Campbell, A. M. Critchley, C. R. Croft, C. D. Evans, J. C. Hawksley, R. Johnston, P. J. Jory, D.S.O., J. M. Matheson, T. Parr, T. McK. Robb, J. R. Robertson, E. J. Selby, and W. L. Spencer-Cox, M.C.; Capts. (Temp. Majors) (acting Lieut.-Cols.) S. Brown (killed in action), and P. C. Mayo; Major (acting Lieut.-Col.) D H. Young and Major W. H. Milligan; Capts. (Temp. Majors) J. H. L. Easton, T. J. Guinan, T. G. S. James, P. H. Jobson, R. L. Macpherson, R. J. Millbank, and P. D. Stewart; Capt. (acting Major) S. A. Jenkins; Capts. J. P. Baird, S. H. Campbell, W. G. Graham, A. P. Grant, J. N. T. Hutton, D. H. Jones, M. W. Lloyd-Owen, H. K. Lucas, D. D. Muir, C. F. Murison, R. L. Orchardson, W. D. B. Pettigrew, D. B. Ramsay, E. R. B. Reynolds, D. R. Sandison, M. A. Shammas, H. G. Skinner, F. M. Smith, M.C., G. A. Stephen, J. W. M. Sutherland, D. J. Waterston, M.B.E., D. R. P. Wilkie, C. A. Young, and C. F. Young, M.C.; Lieut. (Temp. Capt.) T. Wright, R.A.M.C.; Capts. (Temp. Majors) C. H. Bliss, P. T. Joseph, and M. Sarwar; Capts. K. Partap Singh and S. C. P. Sinha; and Lieut. D. S. B. Stephens, I.M.S.; Subadar-Major K. S. Bedi, Bahadur, O.B.I., Subadar M. Singh, and Jemadar C. Hans, I.M.D.

CASUALTIES IN THE MEDICAL SERVICES

Died.—Lieut.-Col. A. M. V. Hesterlow, I.M.S.

Wounded.—War Subs. Col. E. C. Beddows, M.C., Temp. Lieut.-Col. W. D. F. Lytle, War Subs. Capt. T. P. W. McCarthy, R.A.M.C.

DEATHS IN THE SERVICES

By the death of Lieut.-Col. ROGER PARKER WILSON, C.I.E., I.M.S., on Dec. 12 at Southport the Indian Medical Service loses a distinguished and popular member. He was educated at University College, Liverpool, and St. Bartholomew's Hospital, London, passed the Conjoint finals in 1893, and took a good place at the entrance examination to the I.M.S. in 1896. Later he obtained the Cambridge D.P.H. in 1900 and the F.R.C.S.Eng. in 1912 while on leave. He served with the Indian Army until 1903, and was then appointed to the Bengal Civil Medical Service. After holding posts as civil surgeon in various districts he went to Calcutta to the important appointment of superintendent of the Campbell Medical School from 1913 to 1918, when he was promoted to be surgeon to Calcutta Medical College Hospital and successively professor of clinical surgery and professor of surgery in the Medical College. There he found scope for his abilities both professional and administrative, including examinerships, and as president of the Bengal State Medical Faculty and Council of Medical Registration. This led to his acting as Surgeon-General to the Government of Bengal in 1922 and 1925, and he was unfortunate in not obtaining a permanent administrative post before being precluded from further promotion by the age retiring rules. His various services were rewarded by the C.I.E. in 1925 and by his appointment as Honorary Surgeon to H.E. the Viceroy in 1920. He was joint author, with Rogers, of a paper on two cases of liver abscess treated by aspiration after early diagnosis by the blood changes—the first trial of the method now almost universally adopted. (*B.M.J.*, June 6, 1906.) Wilson was a most loyal colleague and was much liked by all his colleagues. In his young days he was a fine athlete and a good football player. Latterly he was much crippled by illness, but never lost his cheerfulness; he will be much missed by his many friends.

Lieut.-Col. JOHN LEWIS MACRAE, I.M.S.(ret.), died at Stornoway on Nov. 25, aged 75. He was educated at Blairlodge School and the University of Edinburgh, where he qualified M.B., C.M. in 1890. He entered the I.M.S. in 1891, and after military service in Burma and with the Peking Relief Expedition he became a district medical officer in the Madras Presidency and was stationed at Mercara as civil surgeon of Coorg for many years. He retired in 1921.

Universities and Colleges

UNIVERSITY OF GLASGOW

A graduation ceremony was held on Jan. 15, when the following medical degrees were conferred:

M.D.—[1]K. S. Alstead, [1]J. Black, [2]A. L. Goodall, A. C. Penman.

PH.D.—(In the Faculty of Medicine): Albert Sharman, M.D.

M.B., CH.B.—R. McL. Archibald, D. Beaton, Ellen S. Bell, G. M. Burns, R. Burns, C. Cameron-Mowat, K. D. Cochran, L. N. Cook, Margaret P. Deighan, Ninian C. Dick, Mary C. Douglas, A. M. S. Elfehfah, Mararet J. W. Finlayson, J. Fraser, T. M. Gardner, Mary E. Hamilton, E. W. Hutcheson, P. McCulloch, D. A. MacDonald, W. G. McEwen, T. S. Macfarlane, M. M. Macgregor, J. Malachlan, R. W. McIkle, R. Meldrum, Katherine G. Miller, M. S. Miller, R. F. Millet, R Mitchell, Alexandrina Moffat, Lilias McA. Money, B. Moscow, Monica M. A. Murphy, W. L. Newbigging, Hetty B. Ockrim, W. G. Paterson, H. A. Rankin, F. E. Roche, L. S. Scott, M. K. M. Scrimgeour, J. Shapiro, Catherine G. Simpson, J. M. Sinclair, Margaret F. Smith, D. J. Stewart, E. McG. Stirling, Agnes C. Sutherland, R. Thomson, J. D. Wilson, Marjorie B. Wilson, J. D. Wimbs.

[1] With high commendation. [2] With commendation.

Medical Notes in Parliament

White Paper on Social Security

On Jan. 18 Mr. CHURCHILL, replying to Mr. Graham White, said he hoped that the White Paper on Social Security would be ready about the end of February or early March, but he could not pledge himself to any particular date.

Awards to R.A.M.C.

On Jan. 18 Sir JAMES GRIGG told Sir Harold Webbe that 204 awards and decorations other than for cases of conspicuous gallantry had been made to R.A.M.C. officers, and 22 to R.A.M.C. other ranks. In addition 499 officers and 381 other ranks had been mentioned in dispatches. No citations accompanied recommendations for mentions, and it was impossible to say how many of them were for cases other than gallantry.

University Grants Committee

On Jan. 18 Sir JOHN ANDERSON informed Mr. Harvey that no change had been made in the terms of reference of the University Grants Committee, which were: "To inquire into the financial needs of university education in the United Kingdom, and to advise the Government as to the application of any grants that may be made by Parliament towards meeting them." It seemed to him that these terms of reference were wide enough to enable the committee to report on any problem on which the Government might need advice in connexion with the making of grants to universities. There had grown up between the committee and the universities an accepted custom of constant informal consultation on matters of university policy, and it was contemplated that this would be continued and developed. Mr. HARVEY asked if there was any possibility of the formation of a university council in the not distant future to make that consultation more effective, but Sir JOHN ANDERSON replied that he could not say anything about that.

Registrar-General's Health Statistics

On Jan. 18 Major MARKHAM asked the Minister of Health whether he was aware that no detailed health statistics had been published for several years; and if he would give authority for the issue of up-to-date civil and medical tables or statistical reviews to supplement the summary reports of the Ministry of Health. Mr. WILLINK said that the Medical Section (Part I) of the Registrar-General's Statistical Review for 1940 was published last September and the Civil Section (Part II) of the 1939 Review last January. Corresponding volumes for subsequent years were being issued as soon as possible; but their production was conditioned by the very restricted printing facilities now available for this class of matter. During the present extreme shortage of paper the volumes could not be placed on public sale, but the statistics might be consulted at the Office of the Registrar-General, from whom a temporary loan of any volumes desired could be had by those having occasion to refer to them.

Medical Recruitment in East Sussex

On Jan. 18 Rear-Admiral BEAMISH asked the Minister of Health if he was aware that the Central Medical War Committee was working a scheme with his Ministry which ignored the representations of the Local Medical War Committees and other committees concerned in East Sussex and, by designating too many local doctors for military service, was causing overwork and anxiety for those concerned with the health of the local population; and if he would adjust the situation. Mr. WILLINK replied that Admiral Beamish was no doubt referring to a plan which he had asked the Central Medical War Committee to put into operation on the recommendation of the Medical Personnel (Priority) Committee as one of the steps to obtain the number of doctors which the Government had decided to assign to the Forces. The plan applied to any area which had not satisfied the quota of doctors required of it—that was to say, which retained a higher proportion of general practitioners to population than that normally adopted for the purpose of calculating the quota. The Central Medical War Committee issued recruitment notices to doctors of military age up to the number required to make up the quota, but it was open to the Local Medical War Committee, as well as to the doctors themselves and any employing authorities, to make representations to the Central Committee against the recruitment. These representations were then considered fully by the Central Committee, at an oral hearing if so desired, before a final decision was reached.

Medical News

Prof. B. A. McSwiney, Sc.D., M.B., will deliver the Oliver-Sharpey Lectures before the Royal College of Physicians of London on Thursdays, March 2 and 9, at 4 p.m., at the College, Pall Mall East. His subject is "Afferent Fibres of the Abdominal Viscera."

At the meeting of the Medical Society for the Study of Venereal Diseases to be held at 11, Chandos Street, Cavendish Square, W., to-day (Saturday, Jan. 29) at 2.30 p.m., a general discussion on "The Intensive Treatment of Early Syphilis" will be opened by Lieut.-Col. D. M. Pillsbury, senior consultant in dermatology, U.S. Army, in association with other American medical officers. At the meeting on Feb. 26 Col. J. E. Gordon of the U.S. Army Medical Corps will give an address on "The Prophylaxis of Venereal Disease."

The Friday evening discourses to be given at 5 o'clock at the Royal Institution, 21, Albemarle Street, W.1, include one on Brain Rhythms by Prof. E. D. Adrian, M.D., F.R.S., on Feb. 4; one on the Medical and Surgical Achievement of Soviet Russia in War by Mr. E. Rock Carling, F.R.C.S., on Feb. 25; and one on Habit and Evolution by Prof. D. M. S. Watson, D.Sc., F.R.S., on March 10. The following courses of lectures have been announced: Modern Developments in Dairy Science by Prof. H. D. Kay, D.Sc., on Feb. 1 and 8, at 5.15 p.m.; on Fungi and Modern Affairs by J. Ramsbottom, D.Sc., on Feb. 15, 22, and 29, at 5.15 p.m.; on the Mode of Action of Some Vitamins by Prof. A. R. Todd, D.Sc., F.R.S., on March 7 and 14, at 5.15 p.m.; on Chemical Factors in Nervous Effects by Sir Henry Dale, M.D., P.R.S., on March 21 and 28, at 5.15 p.m.; and on Food Fads and Food Fallacies by Sir Jack Drummond, D.Sc., on Feb. 24, March 2 and 9, at 2.30 p.m.

On Wednesday, Feb. 9, at 2.30 p.m., the Nutrition Panel of the Society of Chemical Industry will hold a meeting in the rooms of the Chemical Society, 56, Victoria Street, Westminster, London, S.W.1, when Mr. F. A. Robinson, M.Sc., F.I.C., will read a paper on the vitamin B_2 complex. The speaker will discuss not only the properties and distribution of these vitamins, but also their biological functions so far as they are understood.

On Feb. 10 at 7 p.m. at the House of the Pharmaceutical Society of Great Britain, 17, Bloomsbury Square, London, W.C.1, the Harrison Lectureship Medal will be presented to Mr. Arthur James Ewins, D.Sc., F.R.S. Following the presentation, Dr. Ewins will deliver the Harrison Memorial Lecture on "Progress and Problems of Chemotherapy." The development of chemotherapy in relation to pharmacy will be reviewed and attention drawn to some of the problems in the field of chemotherapy which still await solution. It is hoped that there will be a large attendance of members and friends. Refreshments will be served after the meeting.

Imperial Chemical (Pharmaceuticals) Ltd. ask us to print the following statement: It has recently been brought to our notice that toxic symptoms have occurred following anaesthesia in which trilene has been used with a closed circuit. Pending a full investigation of this matter, which is now being conducted, we would suggest that trilene should not be used with a closed circuit.

The Therapeutic Research Corporation of Great Britain Ltd. has elected the following officers for the year 1944: Chairman, Board of Directors, Mr. H. Jephcott (Glaxo Laboratories Ltd.); Deputy Chairman, Dr. F. H. Carr (British Drug Houses Ltd.). Chairman, Research Panel, Dr. A. J. Ewins (May and Baker Ltd.); Deputy Chairman, Mr. F. A. Robinson (Glaxo Laboratories, Ltd.). The Corporation has appointed Dr. Frank Hartley as secretary to take up duties early in 1944. The new offices of the Corporation are at General Buildings, 99, Aldwych, W.C.2.

Mr. Henry Berry, chairman of the Metropolitan Water Board, in a lecture on London's water supply problems before the Royal Society of Arts on Jan. 19, said that the use of larger quantities of chlorine for purification enabled the capital to escape any typhoid epidemic during the heavy air-raid period. At present all the Board's water supplies, whether from river or well, were chlorinated, but he would not be surprised if after the war the Board and other water authorities gave more consideration to the use of ozone as a purifying agent.

A meeting of the Court of Directors of the Society for Relief of Widows and Orphans of Medical Men was held on Jan. 5, with the president, Dr. R. A. Young, in the chair. A sum of £1,965 was voted to pay the half-yearly grants to the fifty-six widows in receipt of relief. Relief is granted only to the necessitous widows of deceased members, or their orphans if under the age of 16. Membership is open to any registered medical man who at the time of his election is residing within a twenty-mile radius of Charing Cross. Application forms for membership may be obtained from the secretary of the Society, 11, Chandos Street, Cavendish Square, W.1.

On Jan. 1 the Bristol Hospitals Fund (Inc.) and the Bristol Aeroplane Company Employees' Hospital Fund amalgamated and merged their funds and interests. As soon as legal formalities can be completed the Fund will be reconstituted as Bristol Hospitals Amalgamated Fund (Inc.), but meantime the Bristol Hospitals Fund (Inc.) will be legally responsible for the affairs of both the existing funds. Address for all communications: Royal London House, Queen Charlotte Street, Bristol, 1.

The latest Order on the control of liver, the Liver Extract (Regulation of Use) Order, 1944, which came into force on Jan. 8, permits the use of proteolysed extract as well as the injectable extract. The general effect of the Order is that liver extract, including desiccated liver, must not be used except in the treatment of pernicious or other megalocytic anaemias, and then only in the form of the injectable and proteolysed liver extract.

The Leverhulme Trustees invite applications for fellowships and grants in aid of research from British-born senior workers prevented by routine duties or pressure of other work from carrying out research. Forms may be had from the secretary, Dr. L. Haden Guest, M.P., Leverhulme Research Fellowships, Unilever House, Blackfriars, E.C.4. They must be returned by March 1.

The following resolution was adopted by the Council of the Malthusian League on Dec. 18, 1943: "Whereas there is an agitation for increasing the annual number of births in Great Britain; and whereas the Government has issued a White Paper (Cmd. 6358) which states that 700,000 births per annum, being the number annually born at the outbreak of the war, would maintain the present population for ever; and whereas no responsible person has advocated an increase of our present population: therefore the Malthusian League earnestly requests the Government to take no steps whatever which might lead to the increase of the annual number of births, especially among the poorer sections of the community, which are already multiplying very rapidly."

The British Homoeopathic Society has lately become incorporated into the Faculty of Homoeopathy, and an inauguration conference was held in London on Jan. 21 and 22, with the president, Sir John Weir, in the chair.

The issue of the *Indian Medical Gazette* for October, 1943, is a special tuberculosis number.

On Tuesday, Jan. 24, at the London Hospital, Whitechapel, in the presence of Sir John Mann, Capt. H. Brierley, and many distinguished physicians and surgeons, a tablet was unveiled to the memory of four radiographers—Ernest Harnack, Reginald Blackhall, Ernest Wilson, and Harold Suggars—who, to quote the inscription on the tablet, "devoted their lives to healing. Their work in the development of the science of *x* rays cost them their health. This they gladly gave in the service of the London Hospital." Dr. J. H. Sequeira, in a memorial speech, briefly traced the history of *x*-ray development from the time of Roentgen. He then paid tribute to the four pioneers, whose "first thought was of their work and their last of themselves." Ernest Harnack joined the hospital staff as a clerk and later had the advantage of being trained by Dr. (now Viscount) Dawson. He started taking radiographs in 1896 with a primitive apparatus, and later he and Dr. Sequeira were able to use a more elaborate apparatus in the post-mortem room, where films were made to determine the value of the rays in diagnosing chest and abdominal diseases. Harnack was the first member of the hospital staff to suffer from *x*-ray dermatitis, and in 1909 he had to relinquish his work. Despite grave mutilation he lived until 1943, dying at the age of 72. Ernest Wilson, who joined the staff in 1899, contracted dermatitis of the fingers after only one year. He continued at work until 1904, but died of rapid cancer in the following year at the age of 39. Reginald Blackhall, who joined the staff when only 18, was particularly successful in stamping out ringworm among the children of the district. With Dr. Sequeira he carried out experiments with *x*-ray filters. In 1925 he succumbed to cancer at the age of 44. By the time Harold Suggars joined the staff in 1903 some of the risks of *x*-ray exposure were known, and he was the first worker to wear lead-lined gloves and body protection. Nevertheless he developed cancers on the body, face, and eyelid. He became almost totally blind from double cataract and died in 1943, showing amazingly cheerful patience and courage in his misfortune. "While we all admire courage," Dr. Sequeira said, "perhaps the most precious possession of all is that secret strength which shows the human spirit to be indestructible in the face of disaster."

No. 1

INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a summary of Infectious Diseases and Vital Statistics in the British Isles during the week ended Jan. 8.

Figures of Principal Notifiable Diseases for the week and those for the corresponding week last year, for: (a) England and Wales (London included). (b) London (administrative county). (c) Scotland. (d) Eire. (e) Northern Ireland.

Figures of Births and Deaths, and of Deaths recorded under each infectious disease, are for: (a) The 126 great towns in England and Wales (including London). (b) London (administrative county). (c) The 16 principal towns in Scotland. (d) The 13 principal towns in Eire. (e) The 10 principal towns in Northern Ireland.

A dash — denotes no cases; a blank space denotes disease not notifiable or no return available.

Disease	1944					1943 (Corresponding Week)				
	(a)	(b)	(c)	(d)	(e)	(a)	(b)	(c)	(d)	(e)
Cerebrospinal fever ..	92	2	28	12	2	87	3	18	7	3
Deaths		3	1				1	—		
Diphtheria	661	38	179	125	32	841	46	238	89	29
Deaths	22	2	5	5	1	36	1	2	5	1
Dysentery	165	22	37	—	—	119	23	32	—	—
Deaths				—	—				—	—
Encephalitis lethargica, acute	1	—	—	1	—	1	—	—	—	—
Deaths		—					—	—		
Erysipelas			58	14	6		—	63	12	4
Deaths		—					—			
Infective enteritis or diarrhoea under 2 years				11					13	
Deaths	44	11	10	10	3	49	5	7	11	6
Measles	515	82	43	82	3	14,153	698	525	11	17
Deaths	—	—	—	—	—	23	2	1	1	—
Ophthalmia neonatorum	64	6	15	1	—	77	2	25	—	—
Deaths										
Paratyphoid fever ..	4	—	1 (B)	1	—	5	—	3	—	4
Deaths	1 (B)	—	—		—	—	—	—	—	
Pneumonia, influenzal*	1,608	140	79	44	6	1,091	67	14	14	2
Deaths (from influenza)	255	31	21	5	2	65	4	3	1	—
Pneumonia, primary ..			516	28				216	26	2
Deaths		85		28	17		—		27	5
Polio-encephalitis, acute	1	—				1	—			
Deaths		—					—			
Poliomyelitis, acute ..	3	—	3	1	1	2	1	2	10	1
Deaths		—					—			
Puerperal fever		4	12		1	—	1	27	2	1
Deaths		—					—			
Puerperal pyrexia† ..	147	11	13	1	5	125	9	20		—
Deaths										
Relapsing fever ..	—	—			—	—	—			—
Deaths										
Scarlet fever	1,791	108	220	37	87	1,808	134	346	42	52
Deaths	—	—	—	—	—	1	—	—	—	
Smallpox	—	—	—	—	—	—	—	—		—
Deaths				—	—	—	—	—	—	—
Typhoid fever	4	—	2	10	3	8	2	5	7	2
Deaths	—	—	—	—	—	—	—	—	—	—
Typhus fever	—	—	—	—	—	—	—	—	—	—
Deaths				—	—			—	—	—
Whooping-cough ..	1,793	195	121	76	25	1,302	86	76	45	6
Deaths	7	1	2	1	1	11	—	7	1	—
Deaths (0–1 year) ..	425	75	120	61	26	407	39	81	48	33
Infant mortality rate (per 1,000 live births)										
Deaths (excluding still-births)	6,084	977	839	339	162	5,184	774	724	300	141
Annual death rate (per 1,000 persons living)			19·3	22·1	‡			16·3	19·7	‡
Live births	7,001	854	1025	407	281	6,403	761	978	359	298
Annual rate per 1,000 persons living ..			20·8	26·6	‡			20·0	23·6	‡
Stillbirths	228	29	31			266	25	38		
Rate per 1,000 total births (including stillborn)								37		

* Includes primary form for England and Wales, London (administrative county), and Northern Ireland.

† Includes puerperal fever for England and Wales and Eire.

‡ Owing to evacuation schemes and other movements of population, birth and death rates for Northern Ireland are no longer available.

EPIDEMIOLOGICAL NOTES

Discussion of Table

In *England and Wales* during the week the notifications of whooping-cough went up by 284, of diphtheria by 83, of dysentery by 76, and of cerebrospinal fever by 19, but there were 389 fewer cases of acute pneumonia, 238 of scarlet fever, and 31 of measles.

Influenza deaths in the great towns dropped to 255. (For the week ending Jan. 15 the figure is 197.) The largest returns were London 31, Sheffield 13, Liverpool 12.

Scarlet fever was down by 50 cases in Middlesex, by 42 in Lancashire, and by 40 in London; the only rises of note were in Staffordshire 27, and in Durham 22. The rise in diphtheria occurred mainly in the north, especially in Yorks West Riding which recorded 40 more cases than last week, Durham with 34 more, and Lancashire with an increase of 28. Whooping-cough notifications were up by 38 in Essex. Returns for measles were fairly steady, the only variation of note being in London, where the notifications have doubled in a fortnight.

Dysentery notifications were almost twice those of the preceding week. Two of the largest new outbreaks were in Sussex 27 (Bognor Regis U.D. 4 and Chichester R.D. 23) and the Port Health District of Liverpool 13. The other large centres of infection were London 22, Lancashire 22, and Surrey 15.

Notifications of cerebrospinal fever have risen from 53 to 92 during the past two weeks: a rise during the first quarter of the year has in recent years been a feature of this disease.

In *Scotland* whooping-cough notifications were up by 48, measles by 25, and diphtheria by 8, while scarlet fever was down by 36 cases, dysentery by 34, and acute primary pneumonia by 19. One-third of the cases of dysentery were notified in Edinburgh.

In *Eire* there was a general rise in the incidence of infectious diseases—diphtheria by 35, whooping-cough by 33, and scarlet fever by 11 cases. Of the 82 notifications of measles 52 were reported from Dublin C.B. and 17 from Roscommon, Castlerea R.D.

Quarterly Returns

England and Wales.—The birth rate during the third quarter of 1943 was 16.2 per 1,000 and was the highest rate for a September quarter since 1930. Infant mortality was 40 per 1,000 live births, this being the lowest quarterly rate ever recorded, and 4 below the average of the ten preceding third quarters. Stillbirths were 2.9% of the total births registered. Diarrhoea accounted for 998 deaths under 2 years of age, giving a rate of 5.9 per 1,000 live births, compared with 5.9, 5.9, 4.9, and 5.1 in the four preceding quarters. The general death rate was 9.4 per 1,000, compared with 9.3 for the third quarter of 1942, and an average of 10.0 for the September quarters of 1937–41. The natural increase—excess of births over deaths—was 70,839, and the increases during the September quarters of 1940–2 were 37,359, 46,285, and 71,000 respectively. The marriage rate, 15.6 per 1,000, is the lowest rate for a third quarter since 1917: 162,908 persons married in the quarter, 28,518 fewer than in the third quarter of 1942, and 87,947 below the average of the five September quarters before 1942.

Northern Ireland.—During the September quarter the births were equivalent to a rate of 23.8 per 1,000, being 2.7 above the average for the third quarters of 1938–42. Infant mortality was 76 per 1,000 registered births, and was 12 above the average of corresponding quarters of the five preceding years. The general death rate was 11.2 per 1,000, and was 0.3 below the average of September quarters for the preceding five years. The death rate from the principal infectious diseases was 0.75 per 1,000, compared with the five-year average of 0.57. This rise was due to diarrhoea and enteritis under 2 years; 167 deaths were attributed to this cause, against an average of 106. Deaths from pulmonary tuberculosis were 188, and 67 from other forms of tuberculosis; the averages of the five preceding third quarters were 211 and 71.

Week Ending January 15

The returns of the notifications of infectious diseases in England and Wales during the week included: scarlet fever 1,747, whooping-cough 2,023, diphtheria 673, measles 559, acute pneumonia 1,294, cerebrospinal fever 64, dysentery 175, paratyphoid 4, typhoid 1. Influenza deaths in the large towns numbered 197.

F. S. Cheever, B. B. Breese, and H. C. Upham (*Ann. intern. Med.*, 1943, **19**, 602) found that sulphadiazine was effective in clearing the nasopharynx of meningococci in 161 meningococcus carriers receiving 8 g. of the drug over a period of 72 hours. No untoward results of the drug were noted.

Letters, Notes, and Answers

All communications in regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1.

ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.

Authors desiring REPRINTS should communicate with the Secretary of the Journal Board, B.M.A. House, Tavistock Square, W.C.1, on receipt of proofs. Authors over-seas should indicate on MSS. if reprints are required, as proofs are not sent abroad.

ADVERTISEMENTS should be addressed to the Advertisement Manager (hours 9 a.m. to 5 p.m.). Members' subscriptions should be sent to the Secretary of the Association.

TELEPHONE NO.—B.M.A. and *B.M.J.*: EUSTON 2111.

TELEGRAPHIC ADDRESSES.—EDITOR, *Aitiology Westcent, London*; SECRETARY, *Medisecra Westcent, London*. ADVERTISEMENTS, *Articulate Westcent, London*.

B.M.A. SCOTTISH OFFICE: 7, Drumsheugh Gardens, Edinburgh.

ANY QUESTIONS?

Which Sulphonamide?

Q.—*In view of the introduction of new sulphonamides, I should be glad to know which is now considered to be preferable for the treatment of pneumonia due (a) to the pneumococcus, and (b) to other organisms, such as the staphylococcus, streptococcus, Friedländer's bacillus, etc.*

A.—No dogmatic answer to this question is justified. There is neither experimental nor adequate clinical evidence on which to assess the relative merits of the newer sulphonamide compounds, particularly for the less common forms of pneumonia. All are believed to act in the same way, and choice may depend not so much on specificity of effect on a particular micro-organism as on some other and perhaps subsidiary property.

With the exception of sulphanilamide, all compounds in common use have a satisfactory action on the pneumococcus. Sulphapyridine, the first to achieve this, and still the most widely used, has a drawback in its strong tendency to produce nausea and vomiting. This is overcome in sulphadiazine, which achieves much the same therapeutic result with far less discomfort to the patient. Sulphamezathine closely resembles sulphadiazine, and has the further advantage of greater solubility, both of the drug itself and of its acetyl compound: absorption is thus rather more rapid and the deposition of crystals in the urinary tract, causing haematuria or ureteric obstruction, is most unlikely to occur with this drug. It is also rather more slowly excreted, and less frequent dosage will therefore serve.

There is good evidence of the efficacy of sulphapyridine in infections due to *H. influenzae*, of sulphathiazole for staphylococcal infections, and of sulphadiazine for those due to Friedländer's bacillus. This is not to say that other new compounds will not serve equally well. Haemolytic streptococcal infections respond to any of the sulphonamides, including sulphanilamide, but in pneumonia the choice should perhaps fall on one with the least possible tendency to cause nausea or cyanosis, such as sulphadiazine or sulphamezathine.

Menopausal Flushes

Q.—*I should be grateful for some information on the treatment of the hot flushes and sweatings incidental to the menopausal changes. In the case I have in mind the attacks occur at night—often as many as six—and are very distressing.*

A.—The specific treatment is by means of oestrogens. A previous answer on oestrogen therapy for menopausal symptoms (*Journal*, Aug. 7, 1943, p. 190) was, by reason of its brevity, open to some misunderstanding, and attracted some valid criticism from Dr. P. M. F. Bishop in a letter published in the *Journal* on Aug. 21, 1943, p. 244. The original answer and this letter should be consulted. Whatever differences of opinion there may be in other respects, all are agreed that the patient should be given the *smallest* dose of oestrogen which will relieve symptoms, the actual dose employed being a matter of trial in individual cases. Natural or synthetic oestrogens may be employed, the latter being relatively more powerful in their effects, especially when administered orally. In this particular case only a very small dose should be required, and oestrone or oestrone benzoate 0.1 mg. or 0.2 mg. t.d.s. by mouth should be tried in the first place. If this is inadequate it can be cautiously increased, and, if need be, supplemented by a weekly intramuscular injection of 5 mg. oestradiol benzoate. Alternatively, and again if a large dose proves necessary, a synthetic oestrogen such as stilboestrol 0.5 mg. once or twice daily by mouth can be employed. The object should be to ameliorate symptoms rather than relieve them completely. Having arrived at the smallest dose which achieves this object, maintain that dose for two to three weeks, and then gradually reduce over the course of several weeks. By the end of three months it should be possible to discontinue treatment completely. An essential feature of the treatment is the regular and gradual reduction in dosage—thereby allowing the body to readjust itself to the altered endocrine balance. Symptoms will recur if treatment is stopped suddenly. If for any reason oestrogen therapy is contraindicated then a small dose of thyroid, or a large dose of calcium, or vitamin E, is worthy of trial.

"Booster" Doses in Typhus Immunization

Q.—*An antigen prepared from typhus rickettsia grown in the yolk sac of developing chicks is being extensively used for immunization purposes. For the initial inoculation the three graduated doses are given at weekly intervals. Thereafter three-monthly "booster" doses are recommended. Is there in fact any weight of scientific data to back the recommendation that reinoculation should be performed at such very short intervals? I know of no other active immunization procedure which demands four "booster" doses per annum to maintain immunity.*

A.—Immunization against louse-borne typhus is still in the experimental stage. Rickettsial vaccines are now employed on a large scale for the first time, and so far little is known about their prophylactic value in the field. Reports from German sources indicate that inoculation with the available vaccines, including those made from infected yolk sacs, does not prevent subsequent infection, but greatly reduces the severity of the disease. It is not known for how long this state of relative immunity lasts following the three doses that are given in the course of the initial inoculation. The laboratories issuing yolk-sac vaccines recommend reinoculation at various intervals—for instance, every four to six months, or every three to nine months. In view of the high fatality rate recently observed in some localities in cases of louse-borne typhus it seems to be a wise decision to adopt the short intervals for reinoculations so long as there is serious danger of infection. It would not be safe to rely on experiences with other immunization procedures. Rickettsiae occupy a position quite separate from the bacteria and the filterable viruses, and the conditions governing immunity to rickettsial infections may differ in many respects from those obtaining in the other groups of micro-organisms.

The "Stammering Bladder"

Q.—*In his book "Sympathetic Nervous System in Disease" Sir Walter Langdon-Brown refers to the "stammering bladder." I should be glad to know just what the physical explanation of this condition is.*

A.—"Stammering" of the bladder is a neuromuscular incoordination, due to a disturbance of the balance between the sympathetic and parasympathetic innervation to that organ. This results in alternations of precipitate micturition with mild strangury. It is therefore allied to other forms of neuromuscular incoordination.

Retarded Speech

Q.—*A boy aged 2¼, healthy in every respect, can only utter animal sounds. He cannot speak any words, not even "ma" or "ta," although he appears to understand everything that is said to him. What treatment and advice should be given? The family history is quite good. Three other boys, aged 11, 7, and 5, in addition to the mother and father, are all in perfect health. These boys began to talk soon after they passed their first birthdays.*

A.—In all probability this child is either deaf or mentally defective, or has some type of congenital auditory aphasia. Despite the statement that he appears to understand everything, his hearing should be most carefully tested. He may be lip-reading. As regards his mentality more information is needed. Has he passed all his milestones at the proper time? When did he hold up his head?—sit up?—walk? Has he control of his bladder and rectum? If his hearing is normal and there is no evidence of mental retardation, then he may have some form of aphasia for which special coaching by a speech therapist is desirable. But it might be worth while waiting another six months or so, as there are occasional late talkers who suddenly begin to talk normally as late as 3 years or more.

Thyroid Treatment of Cretinism

Q.—*I have a patient, a cretin, aged 22. For two years she had thyroid treatment—between ages 2 and 4—and the mother then stopped the treatment because it made the child too excited. Judging from photographs I should say there had been definite improvement during the period of treatment. Of course she is now a stupid imbecile. Is there any use in trying thyroid now, and if so, what dose do you recommend?*

A.—With cretins who have not received treatment continuously, and who show signs of imbecility, the prognosis is bad, with or without the institution of hormone therapy. The administration of thyroid gland might convert a somewhat docile lethargic imbecile into a very active aggressive and awkward person, with resulting difficulties of management. Nevertheless, I do not consider it

justifiable to withhold thyroid treatment, as beneficial results are not impossible. Should the outcome be unsatisfactory it is always possible to cease thyroid therapy, and after some weeks the condition will probably return to the same state as before. Incidentally, even cretins who have received continuous and adequate therapy from birth or infancy may, after many years, show mental deterioration which is not amenable to hormone therapy. As regards the dosage of thyroid, in this and other cases it is determined by the general response and the pulse rate, but more accurately by the estimation of the basal metabolic rate.

Epididymitis

Q.—*A male patient aged 73, active and working, consulted me five years ago for an acute cystitis and has been under treatment ever since. Bacteriological examination revealed a "free growth of B. coli," and x rays showed the presence of two calculi in his prostate. No pyelitis. He passes urine freely, which contains threads. He was treated with mandelic acid and sulphonamides, with slight amelioration only, until he was put on hexamine, small doses of which make him fairly comfortable. He has now developed an acute epididymitis (second attack). He has arteriosclerosis, feels the cold very much, has persistent backache (relieved by massage), polyuria, and a painful tingling of the soles of his feet—no chilblains. Urine has always been alkaline; prostate not unduly enlarged. A recent report says urine is alkaline, specific gravity 1010, faint cloud of albumin, no sugar, small amount of pus, a small number of red blood cells, moderate deposit of amorphous phosphates, moderate numbers of triple phosphate crystals, a few squamous epithelial cells, large numbers of bacteria, and free growth of B. coli.*

A.—The most urgent trouble at the present moment would appear to be the epididymitis, and even though sulphonamides did not appear of benefit previously, it would be worth while putting him on sulphathiazole now. Recurrent attacks of epididymitis are frequently associated with a fibrous prostate containing calculi, and in one or two cases I have known they were so frequent that I divided the vas in order to cut the path along which the infection travelled. As there appears to be no difficulty of micturition or retention, it is not necessary to operate on the prostate. It is not stated whether the *p*H was estimated when the patient was previously on mandelic acid, and as the urine is persistently alkaline supplementary doses of ammonium chloride would probably be necessary. Mandelic acid treatment is often reported to have failed when its failure is due to a *p*H of 5.5 not having been attained.

Halitosis

Q.—*What suggestions have you for the treatment of halitosis in a woman of 40? Tonsils have been removed, teeth and gums appear quite normal, and she has no indigestion or constipation.*

A.—Fetor oris is due to contamination of the expired air, and, if there is no disease of the nose, mouth, pharynx, or lungs, it is usually caused by food particles which are retained in the mouth and undergo putrefaction. Only occasionally is it the result of volatile chemicals, such as alcohol and acetone, which are excreted into the alveolar air from the circulating blood. It is probably never derived from the stomach except as a momentary effect during belching. The condition should be treated by careful brushing of the teeth and the use of mouth washes after every meal. If there is much furring of the tongue this should be cleaned with a soft toothbrush which should be applied gently and as far back as possible. The best chemical cleaners to use are hypochlorites such as Dakin's solution or chloramine compounds. Between meals the patient may be allowed to suck tablets containing 5 gr. potassium chlorate, but she should be warned of the danger of carrying these carelessly in the pockets as there is a risk of fire from contact with matches.

Decaying Teeth in Children

Q.—*Children, ages ranging from 2 to 8, are often seen with one or more teeth in varying states of discoloration and decay. What principles should guide a doctor in advising filling or extracting in such cases?*

A.—Unless the doctor has the facilities and necessary instruments for excavating the decay from the cavity in the tooth, so as to determine the vitality or otherwise of the pulp, it is indeed difficult for him to advise on this matter, and the decision had better be left to the dental surgeon. If there is no exposure of the pulp then every effort should be made to conserve the tooth in question, but a parent is not likely to approach the doctor unless her child has toothache or some swelling of the face. Bad toothache means that the pulp is already infected, and even with careful dressing and filling the pulp may die and an abscess develop. A dead tooth in a child of this age should always be extracted, as it is sure to become a cause of pain or ill-health. Remember to look out for those small localized abscesses or "gumboils," which may burst and leave behind a sinus without the child complaining. The treatment of early decay will save many hours of pain and unhappiness.

"Luis," "Louis," "Lewis," or "Ludwig"?

Q.—*The angle between the manubrium and the gladiolus is sometimes termed the angle of Luis (?), Louis (?), or Lewis (?). Which is the correct spelling, and who was Luis, Louis, or Lewis?*

A.—The correct spelling is "Louis." Antoine Louis lived from 1723 to 1792. He gave lectures on anatomy in Paris and led the way in the application of medical knowledge to legal problems, taking a hand in various famous cases (Garrison). The German form of the phrase is "Louis'scher Winkel," but the name Louis has sometimes been translated into German as "Ludwig" in this country and elsewhere. Braune stated in 1889 (*Arch. Anat. Entw.*) that he had examined Louis's writings in vain for any reference to this angle, and that the first reference of association with Louis's name occurred in Conradi's book, *Position and Form of Thoracic Organs*, published (in German) at Giessen in 1848. It is probable, however, that the first instance of association would occur not in the author's own writings but in those of someone following shortly afterwards. There was a German anatomist named Ludwig living much earlier (1625–80), but no evidence can be found that his name was ever attached to the angle. The following early works make no reference to Ludwig in this connexion: Cowper (1698), Albinus, *Tables of the Skeleton* (1749), and Cheselden's *Osteographia*. It is probable that Louis's name is correctly associated with the angle.

LETTERS, NOTES, ETC.

Food-poisoning in S. Devon

Dr. J. C. CRUICKSHANK, in reference to the answer to the question on food-poisoning in South Devon in the *Journal* of Jan. 1 (p. 30), points out that the service of the Devon County Laboratory, now supported by its association with the E.P.H.L.S., provides for all practitioners in the county full facilities for laboratory and field investigation of such outbreaks as the one described. It appears that all practitioners in the county should have received the full and informative E.P.H.L.S. Memorandum, No. 2, entitled "The Laboratory Diagnosis of Enteric Fever, Dysentery, and Bacterial Food Poisoning."

Extra Milk for Patients with Influenza

Dr. GUY NEELY writes: During the past few weeks many victims of influenza have needed extra milk. To expect them to rise from their beds—for there is seldom anyone to look after them—still less to travel a mile and a half, wait in a queue at the food office, and take the permit to the food depot is perhaps a little unreasonable even in this bureaucratic age. Why cannot the milk form be given direct to the roundsman, or is this too easy?

Post-herpetic Neuralgia

Dr. E. MILLINGTON (Hove) writes: The answer given to the question about treatment of post-herpetic neuralgia (Jan. 1, p. 31) makes no mention of any form of radiotherapy or physiotherapy, and paints a gloomy picture generally. In many cases complete relief from this pain may be obtained by *x*-ray treatment, even with a history of many years, and other cases are helped by electrotherapy and short-wave treatment. *X*-ray therapy will often alleviate otherwise intractable pain in cases of trigeminal neuralgia, brachial neuritis, and other forms of neuritis, although there is no way of selecting suitable cases beforehand, and some cases will show no response, unfortunately.

Wanted—Cade's "Treatment of Malignant Diseases"

The Anglo-Soviet Medical Council has received a special request from the Soviet Union for a copy of Stanford Cade's *Treatment of Malignant Diseases*. Most copies of the book have been destroyed by enemy action. The Council would therefore be grateful if any reader would be willing to sell his copy. Anyone wishing to do this should communicate with Dr. E. Bunbury, Fountain Hospital, London, S.W.17.

Corrigenda

Dr. J. B. DAVEY wishes to make the following corrections in his letter headed "Colonial Research" printed on Jan. 15 (p. 97): The words "medical work" in line 7 should be preceded by the word "routine"; the name "Ranson" in line 8 should of course read "Manson" (Sir Patrick); and the word "individual" in line 20 should read "individualist."

In line 19 of the first paragraph of the leader on the epidemiology of scarlet fever (Jan. 22, p. 118) the words "over a month" should read "once a month," and in line 14 of the third paragraph the words "these are" should read "there are."